VOLUME 67 • FEBRUARY 1958 • NUMBER 2

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The Development of Stapes Surgery After Five Years
Samuel Rosen

Stapes Mobilization—Problems and Perspectives
Victor Goodhill

Considerations on Mobilization of the Stapes for Otosclerosis
Mervin C. Myerson

Further Development of the Mobilization Operation
Alan Austin Scheer

Radical Surgery on the Temporal Bone and Labyrinth Operation for Hearing Improvement
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The Nature of Senile Changes of the Human Olfactory Bulb and Tract
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Elongated Styloid Process
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Treatment of Complicated Colds with Acellular Bacterial Antigen Complex
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The Adenotonsil Problem
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Postoperative Hemorrhages Related to Atmospheric Changes
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A Short Study of Papilloma of the Larynx
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Papilloma of the Larynx
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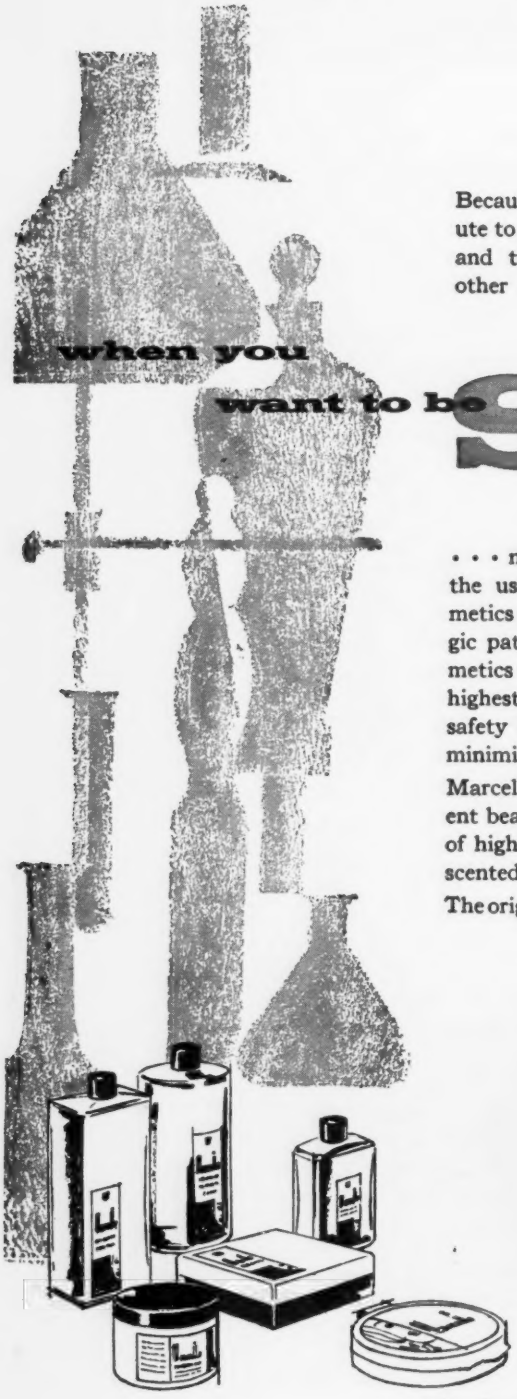

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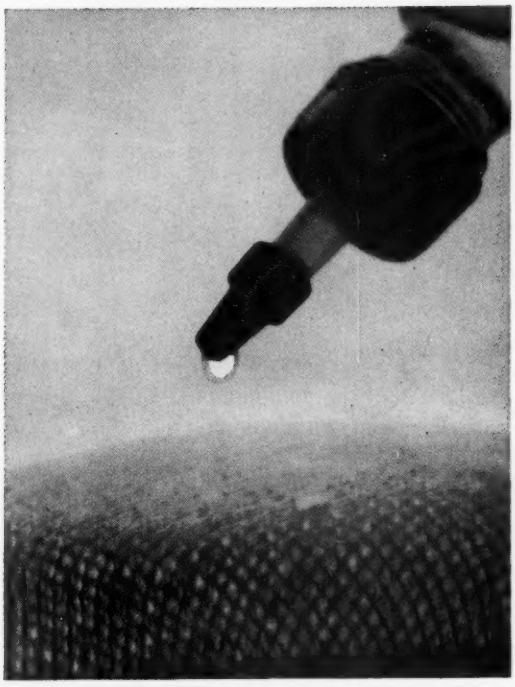

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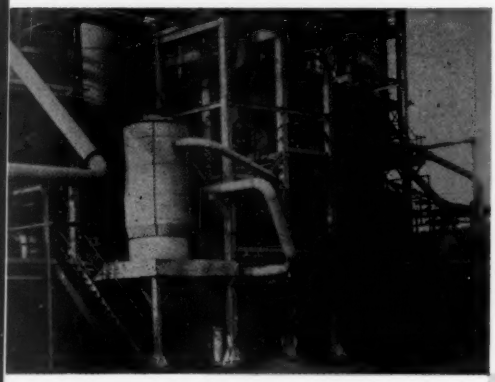

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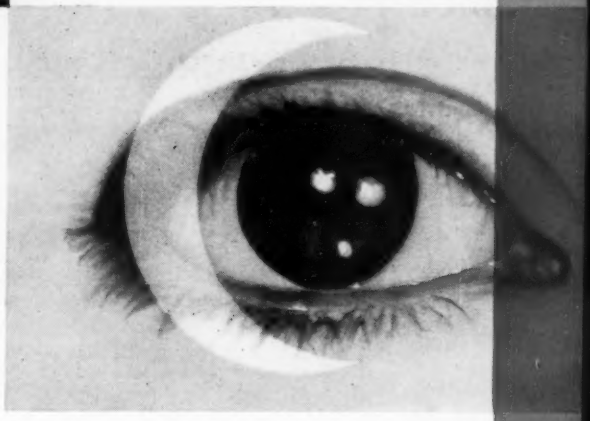

References

1. King, J. H., Jr.; Passmore, J. W.; Skeehan, R. A., Jr., and Weimer, J. R.: Tr. Am. Acad. Ophth. *59*:759, 1955.
2. Kuhn, H. S.: Tr. Am. Acad. Ophth. *55*:431, 1951.

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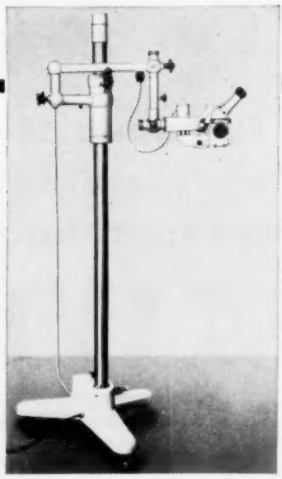

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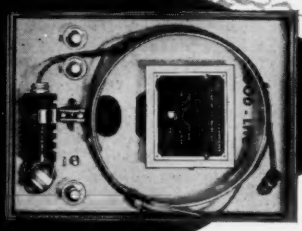

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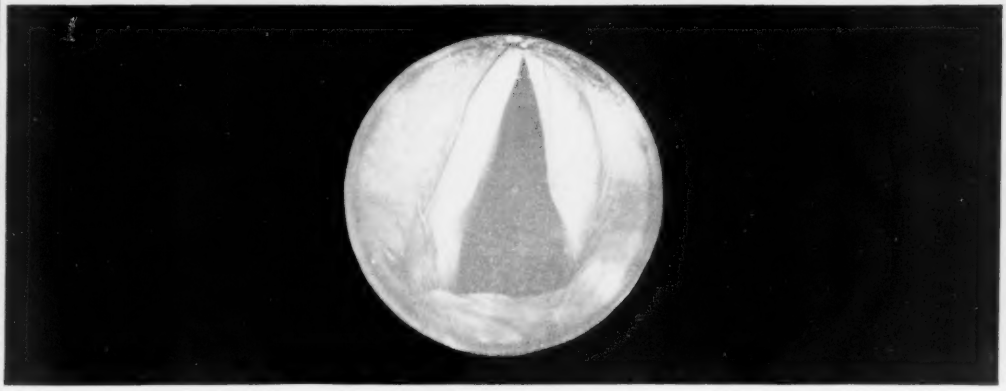

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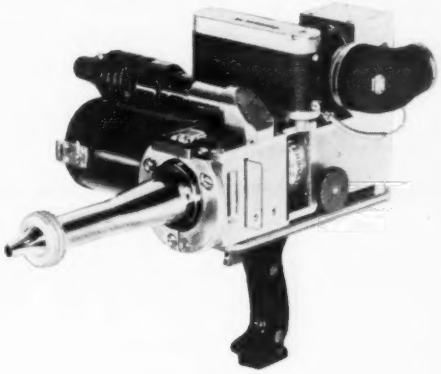

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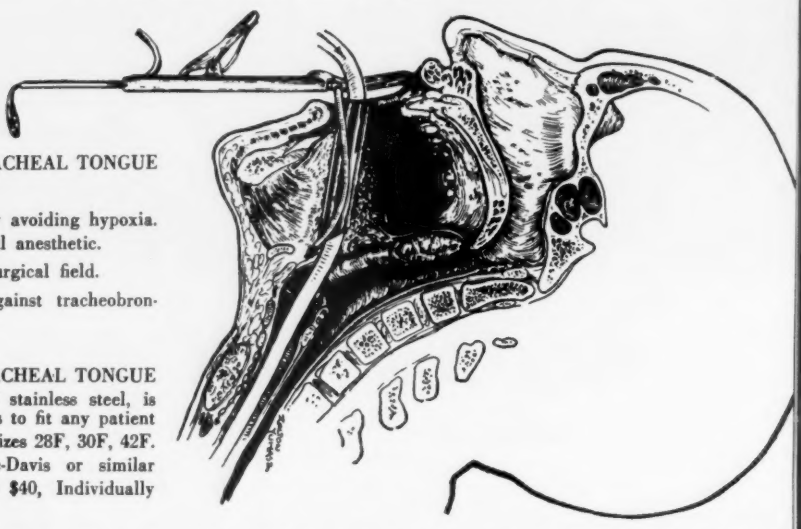

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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The Development of Stapes Surgery After Five Years

Mobilization—Fenestra Ovalis

SAMUEL ROSEN, M.D., New York

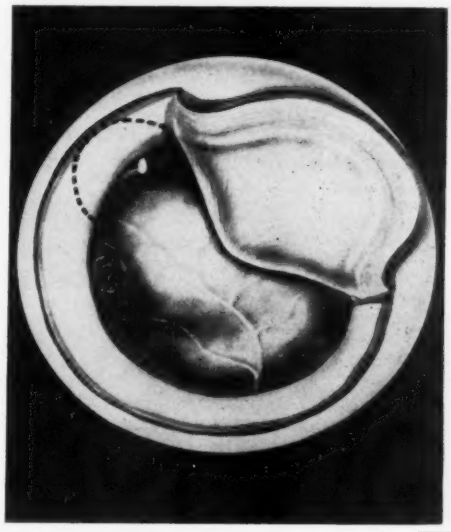
It is now five years since stapes mobilization has been established as a safe and practical procedure for restoring the hearing in otosclerotic deafness. I described my approach to the stapes through the external auditory canal in 1952.[1] Since then, many otologists in America and abroad have used this technique in an estimated 25,000 cases of otosclerosis.

Indirect Mobilization at the Neck

The first technique described [1-10] consists in lifting the drum out of its sulcus and folding it upward upon itself, thus bringing into view most of the middle ear cavity (Fig. 1). Working only through the external auditory canal with an ordinary ear speculum, one removes all of the bone of the posterior canal wall hiding the incus and stapes. With this exposure, the entire long process of the incus, the incudostapedial footplate, facial nerve, and round window can all be seen clearly and in great detail (Fig. 2). The stapes and round window area can then be manipulated with great precision. This approach is in marked contrast to the myringotomy exposure of the last century, which allowed only a haphazard handling of the barely visible stapes and presented serious dangers to the vital structures.[11-24] The stapes operation was abandoned in 1900. However, with the present technique, with better lighting, magnification by loupe and microscope, and the use of antibiotics, stapes mobilization is now a practical procedure.

The hearing has been restored by mobilization at the stapedial neck to various levels,

Fig. 1.—Drum and skin are reflected and most of the middle ear cavity is exposed. However, this does not provide an adequate view of the stapes. The portion of the bone of the posterior canal wall (dotted line) hiding the incus and stapes must be removed in order to permit safe and precise manipulation of the stapes and round window area.



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Consulting Otologist, The Otolaryngological Service, The Mount Sinai Hospital; Head of the Mobilization Clinic, The Mount Sinai Hospital.

Fig. 2.—After 2-3 mm. of the posterior bony canal wall is removed, a full view of the long process of the incus, the incudostapedial junction, the head, neck of the stapes, the entire length of the stapedial tendon, the round window is obtained. This exposure is absolutely necessary for stapes surgery.

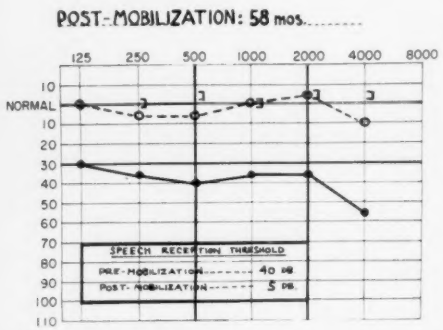


Fig. 3.—An engineer aged 43. A mobilization of stapes was performed in April, 1952, the first case operated on in this manner. Hearing was restored to normal within two weeks after the operation and has been maintained for period shown in this most recent audiogram.

including normal and near-normal. Over 95% of the improved cases have maintained their hearing improvement without regression for periods up to five years. The first patient whose rigid stapes was mobilized on April 3, 1952, and who has been tested repeatedly over the past 58 months, achieved normal hearing two weeks after operation, which has been maintained to date (Audiogram, Fig. 3).

The results obtained with this technique are summarized later in this paper.

Many observers in this country and abroad employing this technique of stapes mobilization have reported results comparable to those reported by me.

Mobilization at the stapedial neck has been referred to as the "indirect" method [25] since the force which is necessary to mobilize the rigid footplate is applied to the neck of the stapes and not to the footplate directly.

Direct Mobilization at the Footplate

The main reason for failure to improve the hearing by the "indirect" method was that in most cases continued pressure on the stapedial neck caused the crura to fracture before the footplate could be mobilized. In other cases, with tremendous force on the stapedial neck, the crura were so strong that they did not fracture, but the hearing did not improve because the footplate remained rigidly fixed in the oval window and could not be mobilized.

Failure to mobilize the footplate by pressure on the stapedial neck led to the second step in the evolution of this operation: going directly to the footplate itself in order to mobilize it. This method was described in 1955 and referred to as the "direct" method of mobilization,[25] since the mobilizing force is applied directly to the periphery of the footplate itself. The sharp-pointed explorer is gently wedged for less than 0.5 mm. into the soft area between the bony rim of the oval window and the periphery of the footplate itself, usually at the inferior margin (Fig. 4). The point of the explorer is moved ever so slightly in and out as if prying loose a lid. When this maneuver is successful, one can suddenly see the entire footplate, tendon, and crura moving. As with the indirect method, this approach has restored the hearing to various levels, including normal and near-normal, by pure tone and speech audiometry (Audiogram, Fig. 5). Improvement of hearing can be obtained whether or not the crura have been

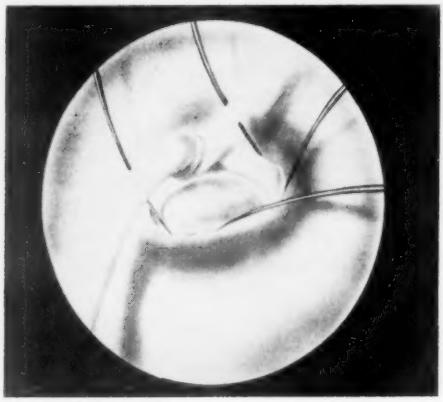


Fig. 4. shows the various sites at which the explorer is used to pry loose the rigid footplate in the direct method of mobilization of the stapes at the footplate itself. The explorer is inserted gently for a distance less than the thickness of the footplate between the anterior aspect of the footplate and the bony rim of the oval window. If the hearing does not improve or the tendon does not yet move, the explorer is then inserted between the inferior aspect of the footplate and the bony rim of the oval window. Much less commonly, it is necessary to insert the point of the explorer at the posterior or superior margins of the footplate in order to mobilize it.

fractured during attempts to mobilize at the stapedial neck.

The results obtained by mobilization directly at the footplate will be summarized later in this paper.

Fenestra Ovalis

When the footplate is extremely rigid, and the otosclerotic process is very extensive, involving both crura, both the indirect (neck) method and the direct (footplate) method of stapes mobilization have failed to improve the hearing. It then seemed logical that in these cases, an opening through the oval window would somehow have to be made to provide for the entry of sound to the cochlea at the site intended by nature. This became the third step in my development of stapes surgery—the creation of a fenestra through the oval window blocked by the rigid footplate—through which sound would presumably be transmitted directly to the cochlea.

It should be emphasized that all attempts at indirect and direct mobilization should

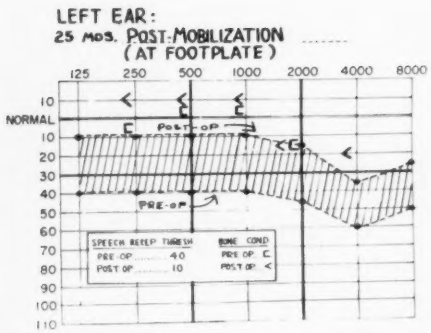
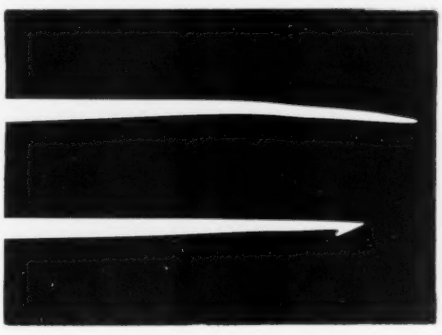


Fig. 5.—A 35-year-old woman with bilateral progressive deafness for more than five years. On March 15, 1954, a mobilization operation was performed on the left rigid stapes by the indirect neck method. The hearing did not improve on the table, and 13 months later hearing tests showed her hearing at the 40-45 db. level. The preoperative hearing level was 55 db. The surgical note at the time of the operation stated that the crura had been fractured. The same ear was reoperated on, April 6, 1955. At the second operation, no technical difficulties or structural changes were encountered in the external canal of the middle ear. Very slight pressure on the stapedial neck revealed free movement of the stapedial tendon, which was probably due to fracture of the crura at the initial operation. Direct footplate mobilization was then performed by inserting the explorer between the bony rim of the oval window and the anterior edge of the footplate, until the footplate was pried loose. The hearing improved on the operating table, and at the last hearing test 25 months later the hearing improvement was still maintained at the 10 db. level.

be exhausted before the fenestration of the oval window is performed.

The technique consists in creating an opening into the vestibule through the rigid footplate of the stapes either with a sharp-pointed explorer or a sharp-hooked instru-

Fig. 6.—The explorer and the hooked oval window fenestrator, used to make a fenestra ovalis through the footplate of the stapes.



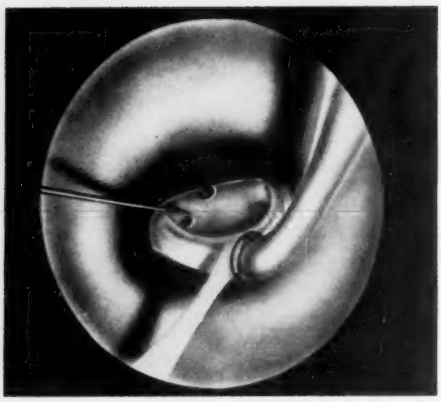


Fig. 7.—The fenestra ovalis is made by inserting the hooked fenestrator into the posterior inferior quadrant of the footplate, either at the periphery or through the body of the footplate as close to the junction of the posterior crus as possible, as shown in this figure. The posterosuperior quadrant may also be used.

ment called the oval window fenestrator. The point of the latter instrument resembles a crochet hook (Fig. 6). When using the oval window fenestrator, one can feel the hook (1 mm. long) catch the footplate and punch out a tiny sliver of bone on its way out after penetration into the vestibule. This is a very delicate maneuver and must be exact.

The site of the penetration in 95% of the cases is in the posterior inferior quadrant of the footplate, either at the periphery or through the body of the footplate as close to the junction of the posterior crus as possible (Fig. 7). This quadrant is usually bluish in color and is the most accessible. The posterosuperior quadrant may also be used. (In many of the cases where the crura had been fractured, the crura and incus were removed in order to visualize accurately the entire naked footplate.)

The physical encounter or contact or collision of the point of the instrument with the footplate must first be *felt* unmistakably before penetration is made. No matter how well one *sees* the contact of the instrument with the footplate, a soft tent-like fibrous layer covering the footplate can often simulate the surface of the footplate to the eye; yet its feel is quite different from that of the bony footplate. Also, the point of the instrument must not only be seen to penetrate the footplate into the vestibule, but it must be felt to penetrate it for not more than 1 mm. This distance can be accurately judged only after extensive kinesthetic practice on the cadaver. This minute procedure must be exact. Since the footplate may vary in thickness from thin cartilage and very thin bone to thick bone, precision must be learned first by practice on at least 100 cadavers. It is also useful to practice the fenestra ovalis several thousand times on substances as nearly like the variable resistances of the footplate as possible, such as an apple, banana, a piece of taut kid as used by eye surgeons, the shell of a small shrimp close to its head, the crust of a hard roll, etc. Fenestration of the oval window should be employed only after considerable experience in mobilization at the stapedial neck and footplate on the living. As in all other stapes procedures, rubber gloves are not used, in order to enhance the tactile and kinesthetic sensations which are essential in stapes surgery. Although the tactile and kinesthetic senses have been found more reliable than the visual, the associated sight and feel are desirable. The point of the instrument should be directed posteriorly, away from the utricle and saccule. After the fenestrator is removed, the opening appears as a discrete hole through which one can see beyond into a dark space—the vestibule. Usually a minimal amount of perilymph can be seen coming through and spreading over the fenestra. When a pledget of cotton is alternately pressed into the round window niche towards the round window membrane, the perilymph can be seen to rise and fall in the created fenestra. Suction should not be applied directly over the fenestra. The fenestra is left uncovered, and the drum and skin are replaced as in the mobilization technique.

Experience has demonstrated that in some cases a fenestra made with the explorer is often too small, since the hearing does not improve on the operating table. But when

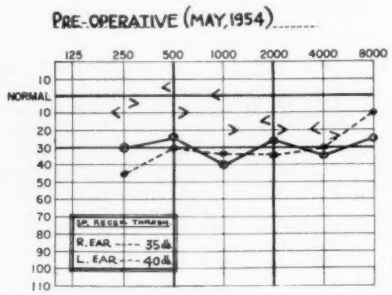


Fig. 8.—A 41-year-old woman with a history of bilateral progressive deafness and tinnitus of seven years' duration. Audiogram 1, taken in May, 1954, shows preoperative air- and bone-conduction and speech reception threshold for both ears.

this inadequate opening is immediately made larger with the hooked fenestrator, the hearing is restored up to levels of normal and near-normal before and after the drum is replaced.

During the operation when the fenestra is made, the patient usually has mild vertigo, sometimes none. After the operation, vertigo may be mild for a few hours to a few days. It is rare that vertigo is severe for more than a few days.

In over 200 fenestrations of the oval window, there has not been a single instance of labyrinthitis or involvement of the facial nerve.

In the beginning when mobilization at the neck of the stapes was the only available technique, stapes mobilization was referred

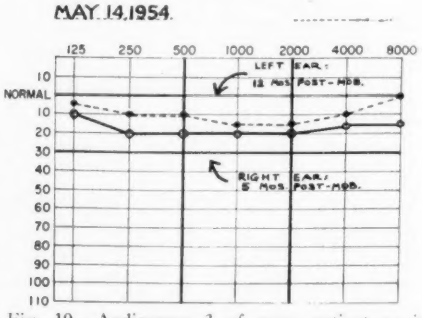


Fig. 10.—Audiogram 3 of same patient as in Figures 8 and 9. On Dec. 2, 1954, a stapes mobilization was performed on the right ear by the indirect neck method. This audiogram shows left ear air conduction one year after operation and air conduction of the right ear five months after operation.

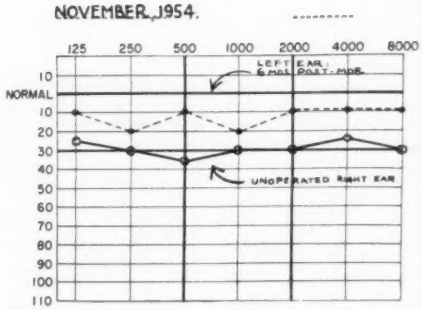


Fig. 9.—Audiogram 2 of same patient as in Figure 8. In May, 1954, a mobilization of stapes was performed on the left ear by the indirect neck method. This audiogram, made six months after operation, shows improved hearing in left ear by air conduction.

to as "a deceptively simple operation but not easy to learn."[10] In the light of the further developments of mobilization at the footplate itself and fenestration of the oval window through the rigid footplate, stapes surgery can no longer be referred to in any way as "simple." It is complex and extremely difficult to perform with adequate skill. Stapes surgery is not nor can it become a standard procedure because each stapes, in its structure, position, anatomical relations, and pathological involvement, is individual and unique. It must therefore be dealt with differently in every case. In one sense, the stapes surgeon, like the custom tailor, must vary his technique to fit each individual.

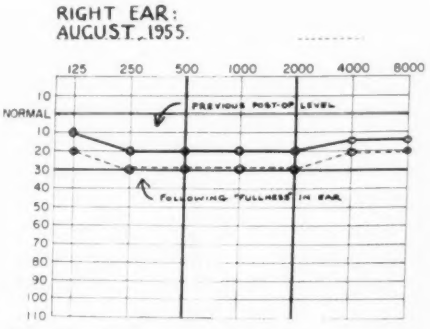


Fig. 11.—Audiogram 4 of same patient as in Figures 8, 9, and 10 shows the right ear only, eight months after mobilization at the stapedial neck. The patient reported a sensation of "fullness" in the ear, and the hearing dropped from the previous postoperative level (indicated by the solid line) to the level indicated by the dotted line.

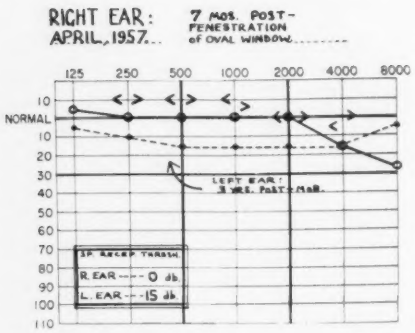


Fig. 12.—Audiogram 5 of same patient as in Figures 8, 9, 10, and 11. On Sept. 12, 1956, this patient's right ear was reoperated on. The crura seemed to have been slightly fractured at the initial mobilization at the stapedial neck. Penetration was made through the body of the footplate with the hooked fenestrator, and a fenestra of the oval window was created. The patient heard on the operating table. Two weeks later the speech reception threshold was at 20 db, from a preoperative level of 35 db. This audiogram, seven months after fenestration of the oval window on the right ear and three years after mobilization on the left ear, shows air and bone conduction and speech reception threshold for both ears. The patient has normal hearing on the right side following fenestra ovalis technique and nearly normal on the left ear.

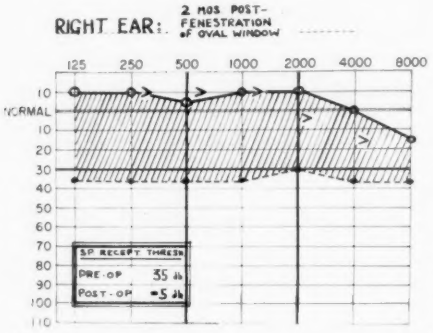


Fig. 13.—A 38-year-old man with bilateral progressive deafness for nearly five years. On Feb. 27, 1957, the right stapes was found to be extremely rigid at operation. A fenestra ovalis was created through the footplate, and the patient heard on the table. The crura remained intact, but the incus was removed to obtain a better view of the footplate, and the stapes footplate was as rigid after the fenestra was created as before. Two weeks after operation, the speech reception threshold was at the 10 db. level. This audiogram shows normal hearing two months after fenestration of the oval window.

Despite interruption of the ossicular chain by fracture and sometimes removal of the crura, the hearing has been improved after fenestration of the oval window to various levels, including normal and near-normal, as measured by pure-tone and speech audiometry (Audiograms, Figs. 8-12).

On the other hand, normal hearing, with complete closure of the air-bone gap, has been attained following fenestration of the oval window where the crura remained intact and the entire stapes, including the footplate, was as rigid after penetration of the oval window as before. The incus was removed before the fenestra ovalis was made in order to get a better view of the stapes. Therefore no sound could possibly be transmitted by vibration of the ossicular chain (Audiogram, Fig. 13).

After the fractured crura and sometimes the incus were removed in some cases, the hearing gain after fenestration of the oval window was considerable with the drum still everted, but the hearing gain disappeared as soon as the drum was replaced. When a myringotomy opening over the stapes region was then made, the hearing gain returned. This problem of the difference in the phase relations of the two windows sometimes works negatively, giving poor hearing when the drum is replaced, whereas in most cases the patient hears as well or better after the drum is replaced.

The mechanism whereby normal hearing has been maintained in many patients for periods as long as 21 months at the latest test following fenestration of the oval window, requires explanation. For the present, however, the empirical facts will have to wait for precise explanation and understanding.

In almost all of the fenestrations of the oval window observed with the loupe and microscope, no evidence of a concomitant mobilization or fracture of the footplate was observed except in one or two cases where only that portion of the footplate which was visible appeared to move as the fenestration was begun. This was also seen only occasionally on the cadaver.

By fenestration of the oval window, normal or near-normal hearing can be obtained

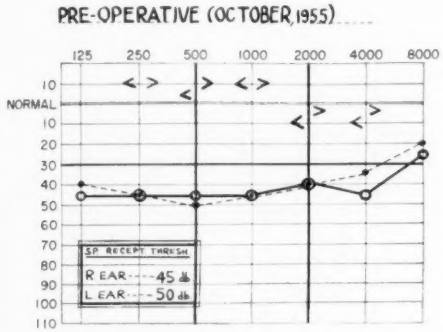


Fig. 14.—A 41-year-old woman with a nine-year history of bilateral progressive deafness and intermittent tinnitus. Audiogram 1 shows the preoperative hearing of this patient.

bilaterally, as in the following case, in which the crura remained intact (Audiograms, Figs. 14-16).

In four cases out of 210 in which this procedure was used, the hearing was made worse by 15 db. below the preoperative level.

Results of Fenestra Ovalis

Of 210 cases in which fenestration of the oval window was attempted, 136 resulted in

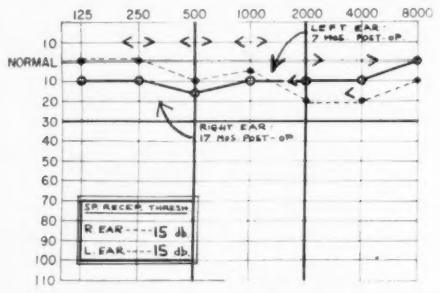
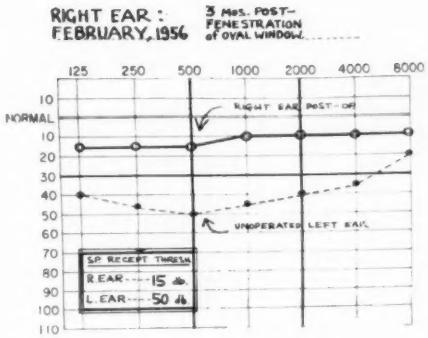

BILATERAL FENESTRATION
of OVAL WINDOW: APRIL, 1957
125 250 500 1000 2000 4000 8000
NORMAL
10 10 20 30 40 50 60 70 80 90 100 110
LEFT EAR: 7 MOS. POST-OP.
RIGHT EAR: 17 MOS. POST-OP.
SP. RECEPT. THRESH.
R EAR----15 db
L EAR----15 db

Fig. 16.—Audiogram 3 of same patient as in Figures 14 and 15. On Sept. 27, 1956, the left ear was operated on. The rigid stapes could not be mobilized at the neck, although the crura did not fracture. A fenestra ovalis was created through the body of the footplate, and the hearing improved. This audiogram, seven months after fenestration of the oval window on the left ear and 17 months after fenestration of the oval window on the right ear, shows the resulting improvement. This patient now has good hearing in both ears and reports that she can now use the phone with the left ear and hear "everything that's going on in the room" with her right ear.


RIGHT EAR: FEBRUARY, 1956
3 Mos. POST-FENESTRATION of OVAL WINDOW
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RIGHT EAR POST-OP.
UNOPERATED LEFT EAR.
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R EAR----15 db.
L. EAR----50 db.

Fig. 15.—Audiogram 2 of same patient as in Figure 14. On Nov. 3, 1955, the right stapes was found extremely rigid at operation, and repeated attempts to mobilize it at the neck failed. The crura remained intact. A fenestra ovalis was created at the periphery of the footplate at its inferior aspect, and the hearing improved on the table. This audiogram shows the improved hearing of the right ear three months after fenestration of the oval window.

the creation of a definite opening into the vestibule which could be seen by the operator and other ear surgeons observing the operation. Further corroboration was obtained when a pledget of cotton was pressed against the round window membrane as described above. In the remaining 74 cases there was no visual assurance that a fenestra had in fact been made, although in some of these cases the hearing showed significant improvement. It was therefore decided to evaluate the results of the fenestra ovalis technique only in those cases in which evidence of a definite fenestra was clear.

The accompanying bar graphs show the results by category in the 136 cases in which the fenestration of the oval window was corroborated at the time of surgery.

The "A" cases were those in which the pure tone loss of hearing by bone conduction was no greater than 10 db. at any of the following frequencies: 500, 1000, and 2000 cps.

The "B" cases include those whose bone-conduction loss was greater than for the "A" cases but did not exceed 20 db. at 500 and 1000 cps and 30 db. at 2000 cps.

The "C" cases were those in which the loss by bone conduction was greater at one

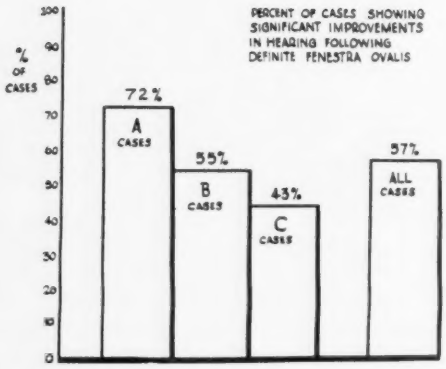

Fig. 17.—Results of 136 cases of fenestration of the oval window, showing improvement of 15 db. or better over the preoperative level by pure-tone and speech audiometry.

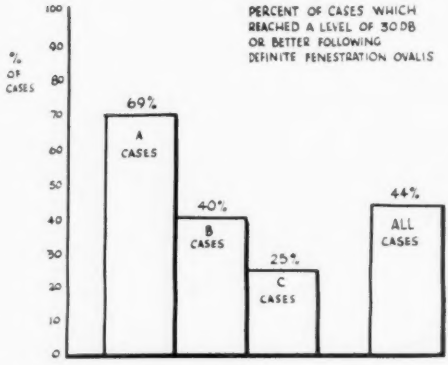

Fig. 18.—Results of 136 cases of fenestration of the oval window which reached the level of 30 db. or better by pure-tone and speech audiometry.

or more frequencies than in the "B" category.

From Figure 17 it can be seen that 72% of the "A" cases, 55% of the "B" cases, and 43% of the "C" cases showed significant improvement in hearing (15 db. or more gain over the preoperative level) following fenestration of the oval window. If the cases in all three categories are combined, thereby including the less suitable with the most suitable, 57% showed such improvement.

Figure 18 is based upon the criterion of hearing restoration to the level of 30 db. or better. It shows that 69% of the "A" cases, 40% of the "B" cases, and 25% of the "C" cases reached these levels, while the combined figure for all three categories was 44%. It is apparent from Figures 17 and 18 that the patients with the better hearing by bone conduction have the highest chances of recovery to useful unaided hearing.

Figure 19 shows that the creation of a new fenestra at the site of the oval window, as described in this paper, has restored hearing to levels of 20 db. or better in 43% of the "A" cases, in 22% of the "B" cases, and even in 11% of the least suitable cases included as category "C." In 26% of all cases operated on in this manner, including cases of varying suitability, hearing was restored to these high levels.

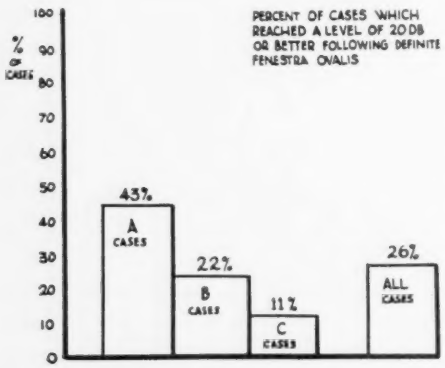

Fig. 19.—Results of 136 cases of fenestration of the oval window, showing improvement to the 20 db. level or better by pure tone and speech audiometry.

Maintenance of Improvement Following Fenestra Ovalis

An important question in every new technique for the surgical relief of deafness is, "Will the improvement last?" I have employed the fenestra ovalis technique during the past two and one-half years. Some of the earliest cases operated on in this manner are shown in the accompanying audiograms with their most recent test data. These demonstrate that hearing improvement following fenestration of the oval window has been maintained for periods up to 21 months at the last test (Audiograms, Figs. 20-27).

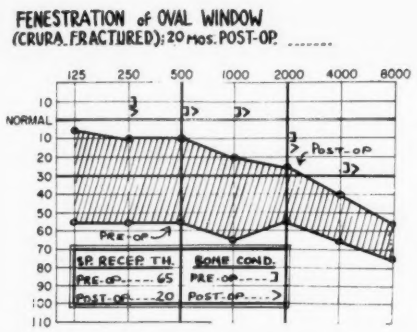

Fig. 20.—A 43-year-old woman with a history of bilateral progressive deafness for 18 years, with constant ringing tinnitus. On Aug. 8, 1955, her right stapes was found to be extremely rigid, and continued pressure against the stapedial neck finally caused both crura to fracture. A fenestra ovalis was made through the center of the footplate, and the hearing improved on the table. This audiogram was made 20 months after fenestration of the oval window.

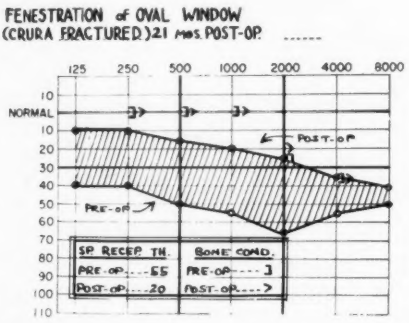

Fig. 22.—A 34-year-old woman with a history of bilateral progressive deafness for 12 years and intermittent tinnitus. On July 19, 1955, the right stapes could not be mobilized by pressure against the neck of the stapes, and the crura fractured. A fenestra ovalis was then made through the footplate. The hearing improved and has been maintained for 21 months as shown in this audiogram.

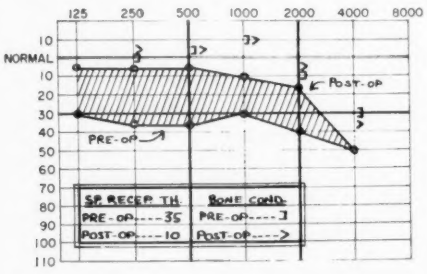

Fig. 24.—A 37-year-old man with bilateral progressive deafness for 18 years and intermittent tinnitus in the left ear. At operation on Aug. 8, 1955, the right stapes could not be rendered mobile by pressure against the neck. The crura remained intact. A fenestra ovalis was created through the footplate, and six weeks later the hearing was at the 10 db. level, which has been maintained for 20 months as shown in this audiogram.

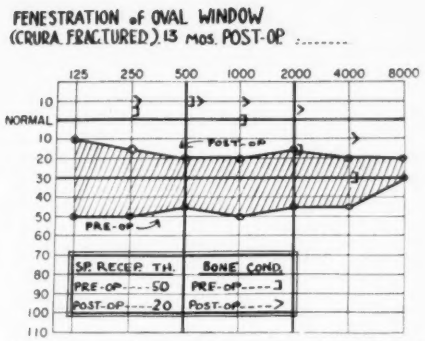

Fig. 21.—A 39-year-old woman with a history of 10 years of progressive bilateral deafness and intermittent tinnitus. On March 2, 1956, her right stapes was found to be extremely rigid at operation, and repeated pressure against the stapedial neck caused crural fracture. A fenestra ovalis was made through the footplate, and the hearing improved and has been maintained for 13 months, as shown in this audiogram.

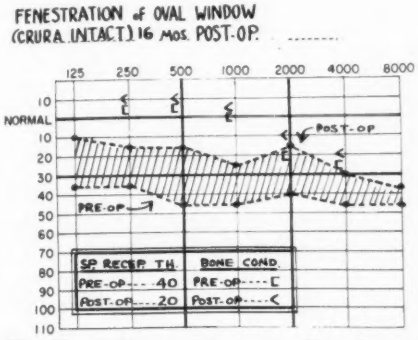

Fig. 23.—A 54-year-old woman with a five-year history of bilateral progressive deafness and constant tinnitus in the left ear. On Dec. 13, 1955, the left stapes was found to be extremely rigid, and great pressure against the neck of the stapes failed to mobilize the footplate. The crura did not fracture. A fenestra ovalis was created through the footplate. The hearing improved and has been maintained for 16 months as shown in her most recent audiogram.

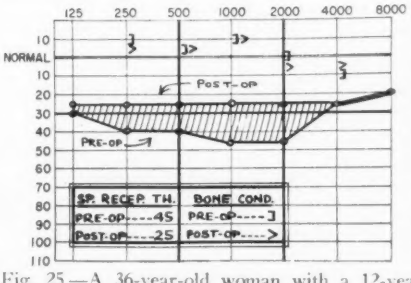

Fig. 25.—A 36-year-old woman with a 12-year history of bilateral progressive deafness and intermittent buzzing tinnitus. The right stapes was found extremely rigid at operation on July 6, 1955, and extreme pressure against the stapedial neck failed to mobilize it. The crura remained intact. A fenestra ovalis was created through the footplate, and the hearing improved. This audiogram shows the maintenance of the improvement for 21 months after operation.

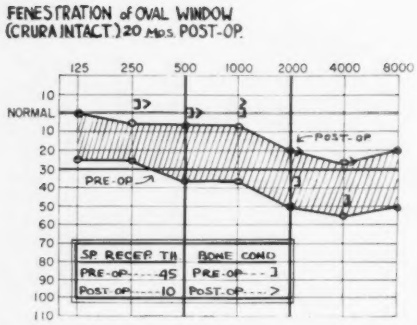

Fig. 26.—A 47-year-old woman with bilateral progressive deafness and constant ringing tinnitus in the right ear for five years. At operation on July 26, 1955, the right stapes was found to be very rigid, and pressure against the neck failed to mobilize the stapes. The crura remained intact. A fenestra ovalis was made by penetrating through the body of the footplate, and the hearing improved. This most recent audiogram shows the maintenance of the improvement for 20 months.

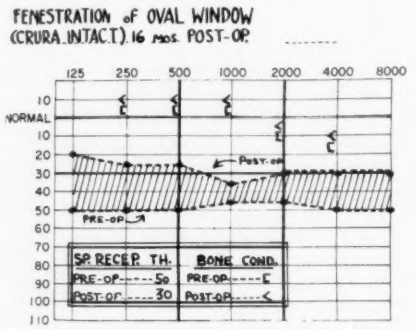

Fig. 27.—A 35-year-old woman with a 15-year history of bilateral progressive deafness with intermittent tinnitus. At operation on Dec. 15, 1955, the left stapes was found extremely rigid and could not be mobilized by extreme pressure against the stapedial neck. The crura remained intact. A fenestra ovalis was made through the body of the footplate. This patient's most recent audiogram, 16 months after fenestration of the oval window, shows that the hearing improvement has been maintained.

Only 9 of the 77 improved cases in this series regressed to or near the preoperative levels of hearing.

In many cases of fenestration of the oval window, the improvement in hearing is gradual, sometimes taking many months to reach its highest level, at which point it becomes stabilized (Audiograms, Figs. 28 and 29). In other cases the hearing improves gradually, reaching its highest level within six weeks to two months (Audiograms, Figs. 30 and 31).

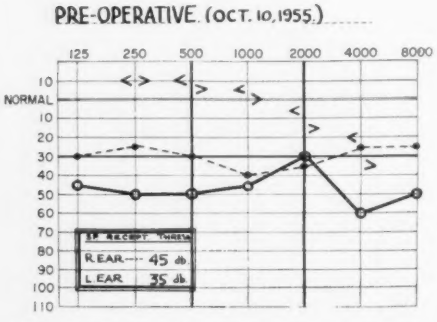

Fig. 28.—Preoperative audiogram of a 42-year-old woman with a history of bilateral progressive deafness and intermittent tinnitus for two years.

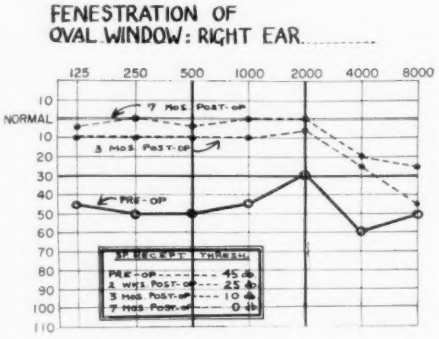

Fig. 29.—Audiogram of same patient as in Figure 28 seven months after operation. This patient was operated on Sept. 11, 1956. The right stapes was found extremely rigid, and the crura fractured during attempts to mobilize at the stapedial neck. A fenestra ovalis was then made by penetrating the body of the footplate. Two weeks after operation the hearing was at the 25 db. level for speech. The interesting aspect of this case is the gradual improvement of hearing following creation of a fenestra ovalis. As the audiogram shows, the hearing continued to improve over a period of months. At one and a half months up to three months, postoperative tests showed the speech reception threshold and average loss by pure tone audiometry at the 10 db. level. When she was tested in April, 1957, seven months after the operation, she was found to have hearing at 0 db. by pure tone and speech audiometry.

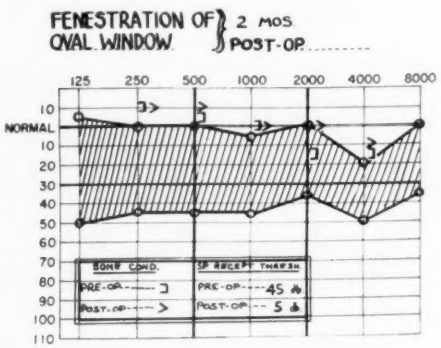

Fig. 30.—A 27-year-old woman with a history of bilateral progressive deafness for four to five years, accompanied by constant tinnitus. At operation on Jan. 11, 1957, the right stapes was found to be very rigid, and repeated pressure against the anterior aspect of the stapedial neck caused the crura to fracture, leaving the rigid footplate. A fenestra of the oval window was then made through the footplate. When tested two weeks postoperatively the hearing was at the 15 db. level for pure tone and speech. As the audiogram shows, two months after operation the hearing was at the 5 db. level for pure tone and speech.

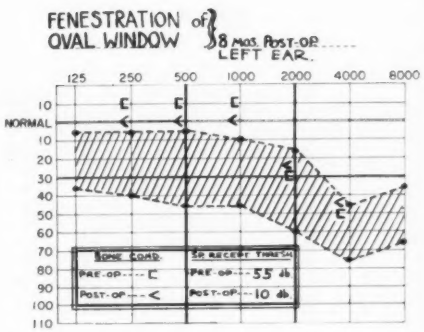

Fig. 31.—A 60-year-old man with a 17-year history of bilateral progressive deafness and intermittent tinnitus. In 1948 another surgeon performed a fenestra nov ovalis on the right ear. The patient wore a hearing aid on the left. On Aug. 7, 1956, after repeated attempts to mobilize the left rigid stapes at the neck failed because of extreme rigidity of the footplate, a fenestra of the oval window was created by penetration through the footplate. The hearing improved on the operating table. One week after operation the hearing reached the 25 db. level for speech and then rose progressively to the 10 db. level for pure tone and speech, which has been maintained as his most recent audiogram shows.

Comparison of Results of the Three Surgical Procedures

Figures 32 and 33 show the comparative results obtained with the procedures developed and reported by me, namely, mobilization at the stapedial neck (indirect method), mobilization at the footplate (direct method), and fenestration of the oval window. Figure 32 shows the per cent of cases in which a significant improvement (15 db. or more gain over the preoperative level) was ob-

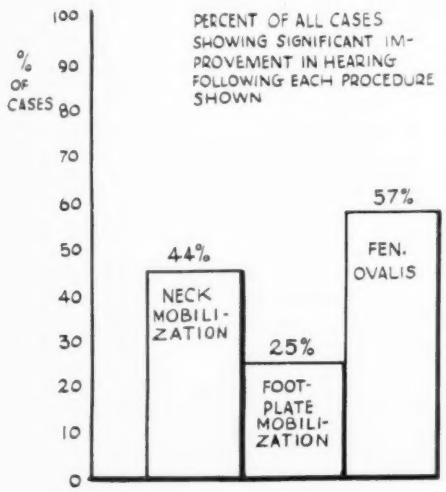

Fig. 32.—Comparative results of each stapes surgery technique for all cases, showing improvement of 15 db. or better over the preoperative level by pure-tone and speech audiometry.

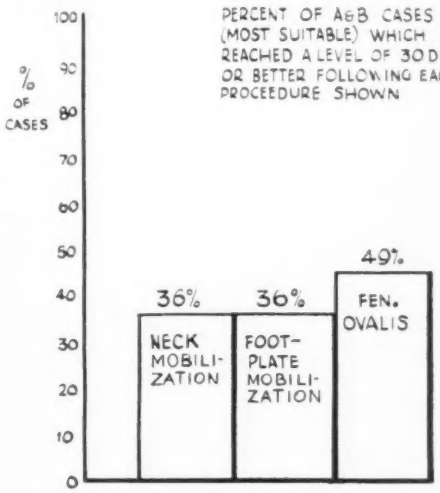

Fig. 33.—Comparative results of each stapes procedure in A and B cases, showing improvement to the 30 db. level or better by pure-tone and speech audiometry.

tained by each procedure. In Figure 33 the results for the more suitable cases (categories "A" and "B") are combined to show the success of each procedure in reaching the 30 db. level or better by pure-tone and speech audiometry.

Over-All Results of Stapes Surgery

A statistical analysis of 254 alphabetically consecutive cases (surnames beginning with A to E) including all cases from the most to the least suitable, revealed that 77% of patients subjected to stapes surgery one or more times by any or all of the above techniques obtained significant improvement to various levels of hearing.

Summary

Three techniques which I have developed for the improvement of hearing in deafness due to otosclerosis are summarized and evaluated.

The detailed results of the fenestra ovalis technique are reported.

When one or more of the described techniques (1, mobilization at the stapedial neck; 2, mobilization at the footplate; 3, fenestra ovalis) was employed, significant hearing improvement was obtained in 77% of all cases operated on.

101 East 73d St. (21).

REFERENCES

1. Rosen, S.: Palpation of Stapes for Fixation, A. M. A. Arch. Otolaryng. 56:610 (Dec.) 1952.

2. Rosen, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis, New York J. Med. 53 (Nov. 15) 1953.

3. Rosen, S.: Simple Method for Restoring Hearing in Otosclerosis: Mobilization of Stapes, Acta-oto-laryng., 44:1, 1954.

4. Rosen, S., and Bergman, M.: Mobilization of the Stapes for Otosclerotic Deafness, read at the Nordic Congress of Oto-Rhino-Laryngology, Helsinki, Finland, June 14, 1954.

5. Rosen, S., and Bergman, M.: Mobilization of the Stapes for Otosclerosic Deafness, Acta otolaryng., Supp. 118, 1954.

6. Rosen, S., and Bergman, M.: Improved Hearing After Mobilization of Stapes in Otosclerotic Deafness, read at the meeting of the French Academy of Oto-Rhino-Laryngology, Oct. 18-21, 1954.

7. Rosen, S., and Bergman, M.: Mobilization of the Stapes for Otosclerotic Deafness: Preliminary Report on 2 Years' Experience, A. M. A. Arch. Otolaryng. 61:197 (Feb.) 1955.

8. Rosen, S., and Bergman, M.: Restoration of Hearing in Otosclerosis by Mobilizing the Stapes Footplate, J. Laryng. & Otol. 59:5 (May) 1955.

9. Rosen, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis, New York J. Med. 55:1 (Jan. 1) 1955.

10. Rosen, S.: Restoration of Hearing in Otosclerosis by Mobilization of the Fixed Stapedial Footplate: An Analysis of Results, Laryngoscope 65:224-258 (April) 1955.

11. Miot, C.: De la mobilisation de l'étrier, Rev. laryng. 10:49, 83, 96, 113, 145, 200, 1890.

12. Boucheron, E.: La mobilisation de l'étrier, Bull. méd. 2:1225, 1888.

13. Kessel, J.: Über das Ausschneiden des Trommelfelles und Mobilisieren des Steigbügels, Arch. Ohrenh. 11:199, 1876.

14. Faraci, G.: Importanza acustica e funzionale della mobilizzazione della staffa; resultati di una nuova serie di operazioni, Arch. ital. otol. 9:209, 1899-1900; also, Tr. Internat. Otol. Cong. (1899), p. 351, 1900.

15. Blake, C. J.: Operation for Removal of the Stapes, Boston M. & S. J. 127:469 and 551, 1892.

16. Blake, C. J.: Middle Ear Operations, Tr. Am. Otol. Soc. 5:306, 1891-1892.

17. Blake, C. J.: Stapedectomy and Other Middle Ear Operations, Tr. Am. Otol. Soc. 5:464, 1893.

18. Jack, F. L.: Removal of the Stapes in Chronic Catarrhal Otitis Media and in Chronic Suppurative Otitis Media, Boston M & S. J. 127:445 and 476, 1892.

19. Jack, F. L.: Remarkable Improvement in Hearing by Removal of Stapes, Tr. Am. Otol. Soc. 5:284, 1891-1892.

20. Jack, F. L.: Further Observations on Removal of Stapes, Tr. Am. Otol. Soc. 5:474, 1893.

21. Jack, F. L.: Further Notes on Removal of Stapes, Boston M. & S. J. 128:8, 1893.

22. Jack, F. L.: Remarks on Stapedectomy, Tr. Am. Otol. Soc. 6:102, 1894.

23. Jack, F. L.: Remarks on Stapedectomy, Boston M. & S. J., 132:34, 1895, footnote 7a.

24. Burnett, C. H.: Partial Myringectomy and Removal of the Incus and Stapes for the Relief of the Lesions of Chronic Catarrhal Otitis Media, M. News, 62:509, 1893.

25. Rosen, S.: Mobilization at the Footplate of the Fixed Stapedial Footplate: Further Development of the Mobilization Technique for Restoration of Hearing in Otosclerotic Deafness, Acta oto-laryng. 45:533 (Sept.-Oct.) 1955.

26. Rosen, S.: Fenestra Ovalis for Otosclerotic Deafness: An Adjunct to Stapes Mobilization, A. M. A. Arch. Otolaryng. 64:227 (Sept.) 1956.

27. Rosen, S.: Fenestration of the Oval Window for Increasing Sound Conduction to the Cochlea, A. M. A. Arch. Otolaryng., 65:217 (March) 1957.

Stapes Mobilization—Problems and Perspectives

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At this stage of stapes mobilization surgery, it appears appropriate to evaluate some existing problems, and to consider some perspectives.

The two problems to be presented are the following:

I. The footplate approach

II. Delayed postoperative threshold shift.

A perspective which merits discussion is the question of combined myringoplasty and stapes mobilization in otosclerotics with tympanic deformities due to coexistent adhesive otitis.

I. The Footplate Approach

There has been a significant evolution in the modus operandi of the surgical technique of stapes mobilization since the first publication of Rosen,[1] when he advised mobilization via the stapedial neck with a specially curved instrument. Shortly thereafter, others[2-3] suggested force application through the lenticular process of the incus, through the capitulum of the stapes, and other modifications in the region of the incudostapedial joint. It was quite logical that initial attempts to mobilize the ankylosed stapedial footplate should begin at the incudo-stapedial joint, because of its anatomic accessibility and the possibility of transmitting force through stable crura. However, it was noted that incudostapedial dislocations could be troublesome (Fig. 1), and that crural fractures (Fig. 2) could be

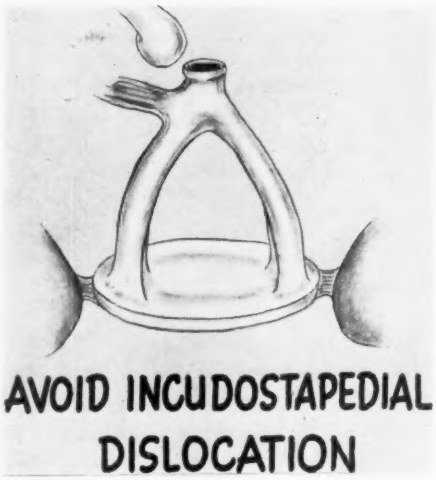

Figure 1

severe problems. In a large number of cases, force applied to the joint itself could give a false sensation of mobilization which in fact was mobility due to crural trauma. In this group of patients the stapedial footplate remained anklyosed even though the stapes appeared to be mobile on digital palpation. This approach, coupled with nonquantitative, nonaudiometric evaluation of adequacy of the surgical procedure, was responsible for many failures.

The recognition of the importance of quantitative measurements, and the utiliza-

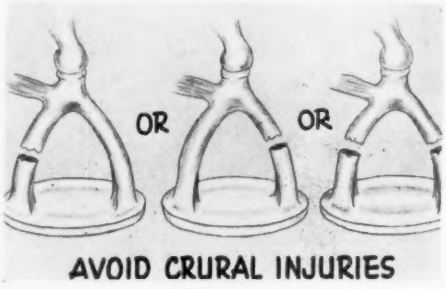

Figure 2

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From the Departments of Otolaryngology, University of Southern California School of Medicine and Cedars of Lebanon Hospital; and the Deafness Research Laboratory at Childrens Hospital, Los Angeles.

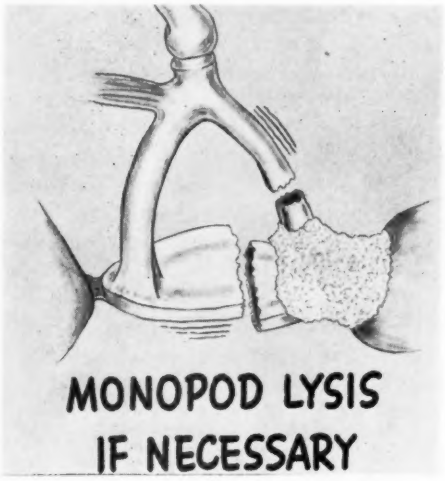

Figure 3

tion of a mathematical formulation of surgical physiology through the audiometric nomograph technique pointed out some of these dangers due to inadequacies in surgical manipulation. This new attitude, coupled with better magnification and illumination of the fossa ovalis and stapedial crura, logically turned the attention of the otologic surgeon to the footplate region.

Footplate attacks have been quite varied and have consisted of direct fractures, incisions, applications of hammer force, fenestration, and peripheral curettage of the footplate region. Each of these procedures has its advocates who ascribe special merit to one or another technique. In all of these footplate approaches, there has been one area of agreement, namely, that preliminary operative results are greatly improved in numbers and in magnitude of threshold shift.

I have previously stated that the purpose of stapes mobilization should be lysis of the ankylosed stapedial footplate in such a manner that the entire footplate or a movable fragment will be in contact with the intact mobile ossicular chain, either through one or both stapedial crura, resulting therefore in either a monopod (Fig. 3) or a bipod (Fig. 4) stapedolysis. This physiological objective remains a necessity.

The creation of a fenestra within the footplate either intentionally or unintentionally by either a crack or a bur hole or a curetted area will result in an opening of the perilymph space of the vestibule to contact the tympanic air space. The exudation of perilymph through such a crack or fenestra will allow the formation of a pool of perilymph in the recessus ovalis. This pool will in effect represent a physiological continuum with the vestibular perilymph space. The square area of such a pool will be at least as large as that of the footplate, and will thus receive a large sound pressure as transmitted through the tympanic air space directly from the speaker's voice or from the audiometric receiver. Thus, a marked improvement in hearing will be realized in almost every case when the footplate is fenestrated (if no intravestibular lesion exists). Such a gain in hearing will be quite comparable with the gain registered by a successful mobilization of the entire stapes in continuity with the ossicular chain and tympanic membrane. It has been claimed that the creation of such a fenestra in the footplate is indeed advantageous and it is recommended as a planned surgical procedure if the footplate cannot be adequately mobilized by pressure through the

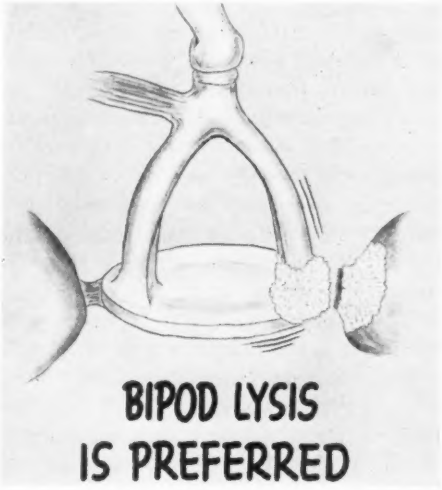

Figure 4

neck, with resultant crural fracture or incudostapedial dislocation.

My experiences in footplate fenestration have been unsatisfactory. While it is true that temporary hearing gains usually occur on the operating table, most of these gains are not maintained, and the vast majority show immediate or early closure. Furthermore, the gains in threshold obtained temporarily by this technique are significantly lower than those obtained primarily by physiologic monopod or bipod stapedolysis.

The utilization of the nomograph technique of surgical audiometry in stapedolysis has been described in detail elsewhere.[4] This technique offers a physiologic guide to stapedolysis surgery in terms of measurement of air-bone gap closure and adequacy of force application. It also is informative in evaluation of the physiologic status of tympanic structures. It is of value, moreover, in the differential diagnosis between a footplate fenestration and a true monopod or bipod stapedolysis procedure. The nomograph technique calls for four operative steps: Step 1 is made after preliminary elevation with the drum closed; Step 2 is made with the drum open prior to mobilization; Step 3 is made after a preliminary mobilization attempt. Steps 3-A, 3-B, 3-C, etc., are progressive steps following progressive mobilization attempts; Step 4 is made after mobilization with the drum closed, and Steps 4-A, 4-B, and 4-C may be necessary in rare instances if inspection of the nomograph reveals a discrepancy suggesting reopening of the middle ear and reassessing the status of the mobilization attempt.

In the event that the force has inadvertently produced a footplate fenestra with a perilymph pool, this will usually show up on the nomograph in the comparison of Steps 3 and 4. In such a case, there will usually be a very adequate gain at Step 3, either directly on the predictive line or to its left. At Step 4, however, in such footplate fenestra, there will be a drop in hearing. If this is demonstrated, a small piece of absorbable gelatin sponge (Gelfoam) is placed in the intercrural space. If such gelfoam packing reverses the relationship and creates a normal rise at Step 4, it will probably indicate that the footplate fenestra existed concomitantly with a mobile monopod or bipod stapedolysis. Should the footplate fenestra exist alone, the absorbable gelatin sponge pack will frequently produce a temporary drop in threshold at Step 3, accompanied by an unchanged level at Step 4. Thus we see a diagnostic value of the nomograph in differentiation between a footplate fenestra and a mobile footplate or footplate fragment (monopod or bipod stapedolysis).

It may be of interest to observe the fate of a significant number of footplate fenestrations that were performed intentionally early in the course of my mobilization experiences. In 1955, a group of more than 50 patients were subjected to footplate fenestration in the hope that this would solve many of the previously insoluble problems. Operative (immediate) successes were indeed high, but postoperative failures were extremely frequent and our over-all average of successes dropped to a very low level during this period. When the approach was redirected to the stapedial circumference and to the area of the stapedial ligament with avoidance of footplate fenestration, there was a marked increase in long-range results.

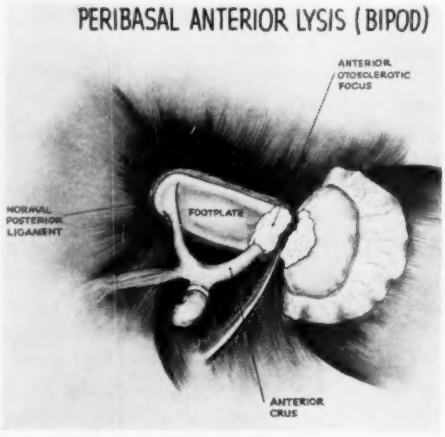

Figure 5

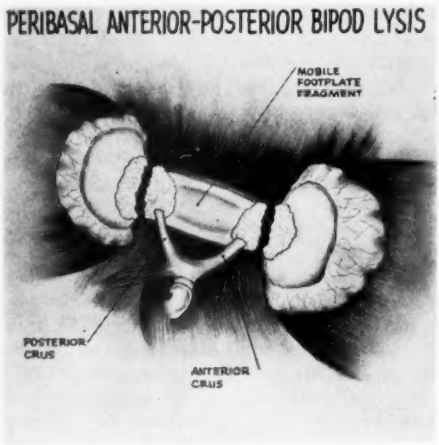

Figure 6

Accordingly, it is my feeling today that in view of our present experience, the locus of choice for force application is the peribasal region (Figs. 5 and 6), i. e., the circumferential or ligamentous area of the footplate in which the calcareous otosclerotic bridge is broken through, and in which attempts are made to preserve inviolate the integrity of the footplate itself without fenestrating it.

Another aspect of the footplate approach worthy of discussion is the increased incidence of vertigo since this approach has been adopted more frequently. The vertigo appears to be utricular in nature, and is immediately noticed on the operating table, in most cases. It may or may not be accompanied by nausea and vomiting, and is usually unaccompanied by nystagmus. It may persist for some time after surgery, although it usually subsides in two or three days, and is greatly alleviated by the use of dimenhydrinate (Dramamine). In a few instances, positional change will elicit vertigo in such patients for many months following stapedolysis surgery, probably owing to otolith displacement because of trauma to the attachment of the utricle within the vestibule. Some patients will have reexcitation of the utricular vertigo postoperatively under the influence of severe exercise involving shaking of the head. The increased incidence of this utricular vertigo has not been discouraging, since the morbidity is not severe and since the footplate peribasal approach has been accompanied by such a significant increase in good results.

II. Delayed Postoperative Threshold Shift

Another problem in stapedolysis surgery is the patient who seems to present no surgical problem in mobilization as ascertained digitally and visually, but nevertheless shows an inadequate gain nomographically. In some cases there may be a marked threshold shift at 500 cps but little or no shift at 1000 or 2000 cps. In other instances, there may be reversal of this frequency relationship, although the former is commoner.

In our experience, this group of patients is small, but nevertheless it is a group of great interest and worthy of analysis.

When the microvibrator or hammer was used on the footplate or on the capitular region in early techniques, it was quite possible that temporary acoustic trauma ensued, with cochlear sequelae producing a resultant lack of threshold shift. In such instances, there may be a recovery phase with a threshold change shortly after surgery. In these cases, the delayed shift in threshold is probably due to acoustic trauma neural hypacusis which is temporary and which is resolved spontaneously.

In an analysis of delayed threshold shift, careful notes at the time of surgery will probably have revealed either a significant incudostapedial joint dislocation or some degree of crural injury, resulting in temporary loss of mechanical coupling. Such coupling may tighten up during the succeeding few weeks, resulting in a marked improvement in hearing.

Excessive tympanic and canal bleeding may cause the accumulation of tympanic blood clots in the middle ear, resulting in a delayed threshold shift. In such instances, there may be a slow but steady return of hearing with closure of the air-bone gap in a matter of two or three months.

III. Combined Stapes Mobilization and Myringoplasty

In those instances where true otosclerosis is accompanied by severe tympanic deformation, otologists in the past have been reluctant to do either fenestrations or stapes mobilizations.

I should like to report a successful technique in the solution of this problem. This has been used in three cases where there has been great deformity and loss of tympanic membrane but an intact ossicular chain and an ankylosed footplate.

The following procedure may be of value in otosclerotic stapes ankylosis, an intact ossicular chain, and a significant drum perforation:

A. Local canal anesthesia by meatal block.

B. Elevation of an extra large omega flap down to and including the fibrous annulus, with exposure of the middle ear. Scar tissue bands in the middle ear are removed and the ossicular chain examined by probe audiometry for evidence of fixation in the incudomalleolar joint. If the ossicular chain is physiologically intact, the tympanic membrane remnant is prepared for myringoplasty. All accumulations of scar tissue and all adhesions within the ossicular chain are removed if possible. Adhesions between the ossicular chain and the tympanic membrane or the promontory are also removed. The round window niche is carefully inspected, and fibrotic lesions in this area are carefully dissected out and removed. The superficial epithelial layer of the tympanic membrane remnant is removed by scraping with a very sharp scalpel, and the margin of the tympanic membrane perforation is removed so that a nonepitheliated margin presents itself to the newly placed graft.

C. The posterior omega skin flap is now completely removed from its attachment to the fibrous annulus. The latter is left in situ. This gives us a large thin full-thickness skin-periosteum graft, which nevertheless is no thicker in most cases than the tympanic membrane itself. This canal graft is carefully put in place temporarily to make sure that it is of the proper size and shape so that it may rest on the deepitheliated tympanic membrane remnant and on the contiguous posterior bony canal wall. Adequate overlapping for proper vascularization is essential. The prepared canal graft is removed and then placed in saline for later utilization.

D. A thin full-thickness postauricular skin graft is removed and prepared and the wound closed with interrupted silk sutures.

E. If probe audiometry has confirmed the diagnosis of a mobile ossicular chain and an ankylosed footplate, the next step is stapedolysis. Peribasal stapedolysis is performed in the usual manner so that ossicular continuity is maintained either in monopod or bipod fashion, preferably bipod. When this has been successfully accomplished as measured by either probe audiometry or nomographic audiometry, the entire tympanic cavity in the region of the open tympanic gap is filled with absorbable gelatin sponge pledgets saturated in Neo-Hydeltrasol solution (a solution containing prednisolone-21-phosphate and neomycin sulfate) to form a bed for the graft. The tympanic graft is then placed in position covering the denuded contiguous drum membrane remnant area and a reasonably generous area of canal wall bone posterosuperiorly and posteroinferiorly. In most instances, this will cause a significant improvement in hearing as measured nomographically, although the operative line may lie somewhat to the right of the predictive line. The postauricular full-thickness skin is trimmed and made as thin as possible and fitted into place in the region of uncovered posterior bony canal wall. The two skin grafts are covered with rayon strips dipped in saline and the canal filled with cellulose sponges dipped in neocortisone ointment. The canal packing is removed on the eighth or ninth day.

Preliminary experiences in three cases yielded excellent results. It seems that this approach may have value in other varieties of tympanic deformations when associated with otosclerotic stapes ankylosis.

As our experiences in stapedolysis surgery accumulate, both old and new problems will require study and analysis. No dogmatic decisions on technique will be permanent. The only unswerving facts are those of underlying tympano vestibular physiology.

It is upon the utilization of basic physiologic laws that perspectives for future problem solutions will be based.

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REFERENCES

1. Rosen, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis, New York J. Med. 53:2650-2653, 1953.

2. Goodhill, V.: Trans-Incudal Stapedolysis for Stapes Mobilization in Otosclerotic Deafness (Under Audiometric Control), Laryngoscope 65: 224-269, 1955.

3. Goodhill, V., and Holcomb, A. L.: A Study of 500 Stapes Mobilizations, Tr. Am. Laryng., Rhinol. & Otol. Soc. to be published, and Laryngoscope 67:615-642 (July) 1957.

4. Goodhill, V., and Holcomb, A. L.: The Surgical Audiometric Nomograph in Stapedolysis (Stapes Mobilization), A. M. A. Arch. Otolaryng. 63:399-410 (April) 1956.

Considerations on Mobilization of the Stapes for Otosclerosis

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Stapes mobilization is the most important present-day interest in otology. Most otologists prefer to ascribe its discontinuance some 50 years ago to the lack of technical advantages. The many reports of success in this field would indicate that mobilization of the stapes will remain as another method for the improvement of hearing in otosclerosis.

Those interested in mobilization of the stapes are trying to establish the procedure on a firm and lasting basis. However, one need only read everything that has been written on the subject since Rosen's first article appeared in 1952, and the literature of preceding years, to appreciate the many unknown or little understood factors which handicap the otologist and influence his results. Some of these are represented in the tremendous variety of histopathologic changes, their extent and location; the significance of the audiogram, the uncertain results, the incidence of recurrence, and an ignorance of the conditions which exist in the middle ear cavity and vestibular structure in a given case. As yet none of these difficulties has been resolved.

In a previous paper the importance of the selection of candidates for stapes mobilization was stressed. It would appear that selection is more important than the technique employed, provided of course, the technique is safe. At the present time, there is no method which enables the otologist to predict marked and sustained improvement in his patients. Therefore, for transossicular stapes mobilization and for mobilization requiring an incision, I prefer cases in which the bone conduction is 15 db. or better in the 500, 1000, and 2000 frequencies, a good air-bone gap, and a lower limit of an air-conduction level of 50 db. Experience has proved that such cases have the best chance for success. This does not mean that cases with poorer conduction levels cannot be improved. They should be given the benefit of any procedure which might help them.

For the past 15 months I have been mobilizing the stapes by transmitting vibrations which originate at the short process of the malleus. In recent months I have substituted an electromagnetic microvibrator and a rod in place of the fork and flattened revolving rod during this procedure. The use of the vibrator simplifies this procedure and appears to attain the objective of mobilization of the stapes with greater facility.

Following is an outline of management of cases selected for stapes mobilization:

Local anesthesia is established by a single injection of 0.5 cc. and not more than 1 cc. of procaine, 1% solution, with 12 minims (0.75 cc.) solution of epinephrine hydrochloride, 1:1000, to the ounce. An excess of solution will obscure the landmarks on the tympanic membrane. A rod, the distal end of which is hollowed conically and connected with an electromagnetic microvibrator, is placed upon the short process of the malleus. The microvibrator is set in motion by a foot switch (Fig. 1). The vibrations so generated are transmitted from the short process through the ossicular chain, to the footplate area. A set screw controls with precision the amplitude of the vibrations. Vibrations are increased to a

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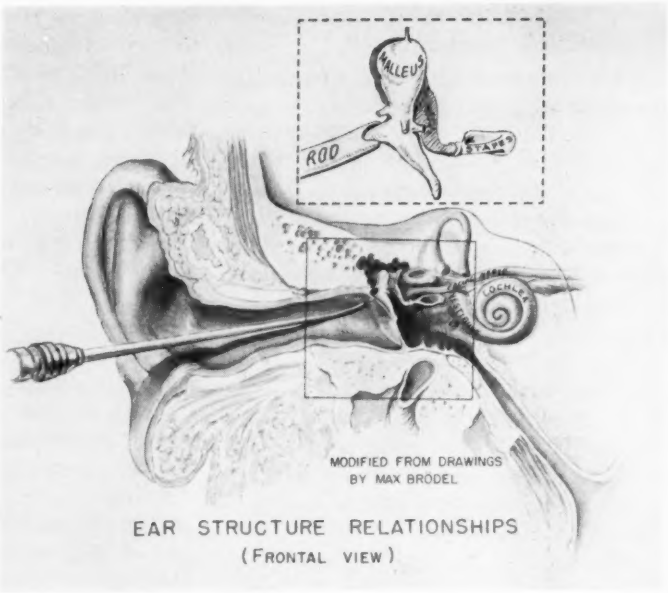

Fig. 1.—Rod attached to vibrator with distal end placed upon the short process of the malleus.

point of tolerance for the individual patient. This limit is reached when pain is experienced.

Trauma in the form of contusion or minor laceration is frequently encountered. Injury to the tympanic membrane in the region of the short process appears to be in direct proportion to the resistance of the ankylosed stapes. When the fixed footplate yields readily to the vibrations and is mobilized, there is frequently no evidence of trauma.

If there is no improvement within a month after this procedure, the open or flap operation is performed. A forked rod attached to the vibrator is placed over the incudostapedial articulation. Vibrations of less intensity than those used in the transossicular procedure are transmitted to the footplate. Here also the amplitude of the vibrations is increased to the point of tolerance (Fig. 2*A*).

When manipulation through the incudostapedial articulation fails, a specially tipped rod, attached to the vibrator, is placed upon the footplate at its inferior border. Vibrations of intensity less than that of those used in the transossicular or transincudostapedial procedures are employed (Fig. 2*B*). Here also the intensity of vibration is increased to the point of tolerance. In four cases which were not improved by the two previous techniques, improvement occurred as a result of vibratory attack upon the rim

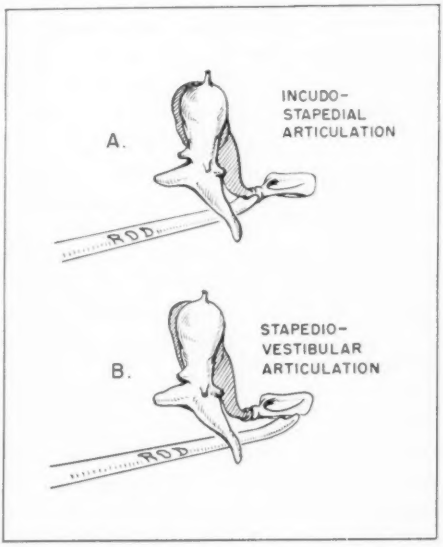

Fig. 2.—*A*, rod in position at incudostapedial articulation. *B*, rod in position at inferior border of stapedial footplate.

of the footplate. In two of these, the improvement was subsequently lost.

Eighty-two cases have been subjected to transossicular mobilization during the past 14 months. Forty-six or 56% of these, were improved. Of those which were not improved, 12 cases were subjected to transincudostapedial vibratory attack. One was improved. In the remaining 11, the footplate was attacked with the vibrating rod, yielding four successes, two of which were subsequently nullified by the recurrence of fixation.

As was mentioned before, the best results obtained are in those cases whose air-conduction level registers better than 50 db. and whose bone-conduction level is 15 db. or better. In addition, a good air-bone conduction gap is improtant.

That the transossicular attack upon the fixed stapedial footplate was thought of long ago is substantiated by the techniques used by several otologists during the latter part of the last century. In 1884, Lucae used a pressure rod which he placed upon the short process of the malleus. It was propelled by a spring. He claimed good results, especially in early cases. Gourivaud (1890) "massaged the tympanic membrane and chain of bones with various instruments and electrical massage." He claimed these were effective. Later, Bellows (1895) introduced a vibrometer which was used several times a week for periods of many months. Wegener (1896) employed an apparatus which he called a vibration masseur, for which he claimed some success. He emphasized that his machine did less for the hearing than for the subjective noises. Harris (1889) reported 19 of 163 cases cured or relieved by an electric masseur designed by Clemens. Large (1899) used a rod attached to a vibrator which was controlled by a 2-volt motor. He claimed good results in the relief of tinnitus and the improvement of hearing. Perhaps the most interesting was the tympanomotor employed by Bonnier (1901). With his technique, the handle of the malleus was grasped and moved in any direction by aspiration through an attached rubber tube. It would appear that, with the exception of Lucae, none of the authors mentioned used sufficient force to obtain best results. It is also significant that a majority employed their devices frequently and over a considerable period of time.

Most otologists of the period just covered were convinced that best results were obtained in early cases in which the Rinne test was positive. Lucae emphasized this point after an experience of 16 years with his pressure sound.

Transossicular stapes mobilization has certain advantages over the operation which requires exposure of the middle ear cavity. Transossicular stapes mobilization, however, should not and does not eliminate the prospect of resorting to other techniques when indicated.

Because of the elimination of the dangers involved in exposure and manipulation within the middle ear cavity and especially at the footplate, the transossicular approach has definite and obvious advantages. The open operation and the fenestration procedure are still available if a good result is not obtained.

In a majority of cases, there is some contusion of the tympanic membrane in the region of the short process. At times there are minor lacerations. These usually heal within a few days. Despite the trauma, immediate improvement of hearing is registered in practically all successful cases.

Transossicular mobilization of the stapes for otosclerosis has proved effective in 50 of 86 patients. Four of the improved cases had chronic adhesive otitis media. These were not included in the cases previously mentioned in this paper. When transossicular mobilization of the stapes fails, other procedures can be employed.

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BIBLIOGRAPHY

Bellows, H. P.: Tabulated Results in the Treatment of Sclerosis of the Middle Ear with the Vibrometer, J. Ophth., Otol. & Laryng. 18: 126, 1895.

Bonnier, P.: Traitement d l'ankylose tympanique, Presse méd. 1:88, 1901.

Goodhill, V.: Trans-Incudal Stapedolysis for Stapes Mobilization in Otosclerotic Deafness, Laryngoscope 65:693, 1955.

Gourivaud, A.: Contribution à l'étude de l'ankylose des osselets de l'ouïe, Toulouse, 1890.

House, H. P.: Personal Experiences with Stapes Mobilization, A. M. A. Arch. Otolaryng. 65: 235, 1957.

Large, S. H.: The Electric Pressure Sound in Ankylosis of Ossicles and Direct Vibrations of Membrana Tympani, Cleveland M. Gaz. 15:499, 1899-1900.

Lucae, A.: Über eine Methode zur mechanischen Behandlung der chronischen Beweglichkeitsstörungen im schallleitenden Apparat des Gehörorgans, Arch. Ohrenh. 21:84, 1884.

Meurman, Y., and Meurman, O.: Stapes Mobilization in Otosclerosis, A. M. A. Arch. Otolaryng. 62:164, 1955.

Myerson, M. C.: Mobilization of the Stapes Without Incision, A. M. A. Arch. Otolaryng. 64: 373-376, 1956.

Experiences with Transossicular Mobilization of the Stapes, A. M. A. Arch. Otolaryng. 66: 480-483, 1957.

Rosen, S., and Bergman, M.: Improved Hearing After Mobilization of the Stapes in Otosclerotic Deafness, J. Laryng. & Otol. 69:297, 1955.

Scheer, A. A.: Observations of 500 Cases of Transtympanic Mobilization of the Stapes, A. M. A. Arch. Otolaryng. 65:245, 1957.

Wegener: Zur mechanischen Behandlung der Sklerose der Paukenschleimhaut, Arch. Ohrenh. 41:199, 1896.

Further Development of the Mobilization Operation

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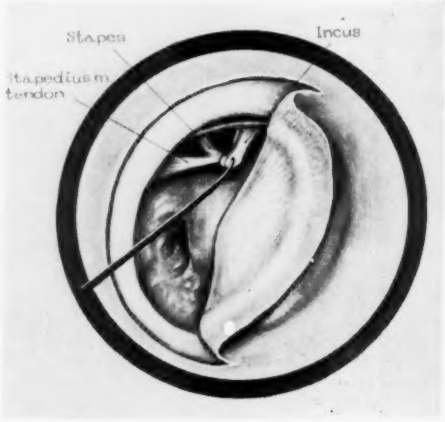
It has been very gratifying to observe the progress that has been made with mobilization surgery. Its reintroduction by Rosen [1] has been the greatest stimulant to otology since the tremendous contributions of Lempert. At the present time, those of us who became interested in mobilization at its revival are aware that there are constant changes in ideas and concepts in this surgery. The knowledge that a fixed stapes could be mobilized has led to the development of varied techniques by the individual surgeons. Rosen's initial concept of applying pressure on the anterior aspect of the neck of the stapes in an effort to mobilize the footplate at first seemed like the logical method to reestablish ossicular conduction. It was quickly determined by others and by me that there were definite limitations in the percentage of good results obtained if this original technique was completely adhered to. Fracturing the crura of the stapes occurred too often to allow the surgeon to feel that he had too great a command over the situation. The outcome of the surgery was too often in the hands of nature and not in those of the surgeons. Goodhill [2] was one of the first to recognize this, and in his first report on the subject he described a varied technique for mobilizing the stapes, which now puts a good deal more control of the outcome of the surgery at the disposal of the surgeon. The concept consists of a step-by-step approach in mobilizing the stapes which will differ in each case according to the degree of fixation. The surgeon must learn to proceed to the next step, should mobilization not be possible, without overstepping that invisible line where that little additional force will fracture the crura.

After 500 cases had been operated on by the standard Rosen technique without any significant improvement in the percentage of good results, the transincudal technique described by Goodhill [2] was adapted and developed. The definite consistent increase in the number of patients that obtained improved hearing was obvious at the start.

The basic difference in the technique has been the change in the direction of the force applied to the stapes, plus the division of mobilizing into separate steps.

Step 1: Pulsating pressure is applied by a curved pick into the end of the long process of the incus. This transmits pressure to the head of the stapes, pushing the footplate into the oval window (Fig. 1). The use of the pick has been described by Goodhill [2] and House.[3] The point of the instrument must be sharp enough to obtain a solid foothold. Too sharp a pick will pene-

Fig. 1.—The point of the pick is set firmly into the end of the long process of the incus, and pulsating pressure is directed towards the footplate.



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trate too deeply into the substance of the incus and may split its long process, or when it is withdrawn may pull it externally and open the incudostapedial joint. Too blunt a pick will slip off the incus, the sudden motion of which will occasionally fracture the crura of the stapes.

The curve of the instrument allows the surgeon to direct the pressure into the incus onto the center of the stapes head or on a line into the posterior crura of the stapes. As I had noted in my original article [4] on this subject, there are three distinct positions of the long process of the incus in relationship to the head of the stapes, which must be noted when using this technique.

1. The incus sets slightly forward on the head of the stapes, with the posterior part of the capitulum visible. This is the most frequent anatomical picture. When this situation occurs, the pick is set forward on the anterior superior aspect of the arm of the incus.

2. The lenticular process of the incus lies flush on the head of the stapes. When this relationship occurs, the pick is inserted in the incus opposite the center of the head of the stapes.

3. The incus sits on the posterior aspect of the stapes head so that its anterior half is exposed to view. This relationship is rare, but when it occurs the blunt pick is inserted directly into the head of the stapes.

In the majority of cases, the incus forms a 90-degree angle with the stapes. If a greater angle is found to exist, the transincudal application cannot be used since the direction of the applied pressure cannot be controlled. A large amount of pulsating force can be transmitted through the head of the stapes down both crura to the footplate. As with all steps in this surgery, instrumentation must be delicate and exact.

When the stapes loosens, there is a sensation of sinking in toward the oval window. If the pressure has been exerted on a line with the posterior crus, there may be a posterior tipping of the stapes at the same time as it moves inwards. As with Rosen's original technique, there is a change in the light reflex on the stapedius tendon.

Step 2: If there is no sign of loosening, then the surgeon proceeds to the next step, where the instrumentation is directly to the

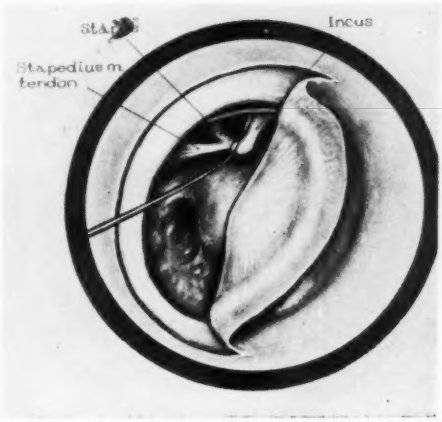

Fig. 2.—The dull point of the pick is set on the visible posterior portion of the head of the stapes. Pressure is directed into the posterior crura.

head of the stapes. Usually there is a lip of the posterior aspect of the stapes head visible behind the incus. It is on this exposed surface that the curved pick is applied, but one with a much duller point to prevent splitting off part of the capitulum (Fig. 2). Pulsating pressure is directed towards the posterior crus. If the transincudal technique has failed, this is frequently effective in accomplishing mobilization.

It is often necessary to alternate this later maneuver with a back-and-forth or rotatory motion, using a sharper instrument placed on the same exposed portion of the head of the stapes.

At the present time, all stapes that are thought to become mobile do result in improved hearing. I had originally reported that only half of the cases, where the stapes was mobilized, obtained improved hearing, but further investigation of many of these earlier cases showed that mobilization had been incomplete or that a fracture of the crura had unknowingly occurred. If there is doubt as to whether there is mobility of the footplate, it can be demonstrated by a change in the light reflex on blood or saline filling the niche of the round window after pressure is applied to the incus.

Step 3: The final step, if the stapes has resisted mobilization, is to approach the

Fig. 3.—A sharp pick is used as a wedge at the margin of the footplate at its anterior border.

footplate directly. This is now a vital part of mobilization surgery if the maximum percentage of good results are to be obtained. The Zeiss operating microscope should be used throughout the entire procedure, but is imperative for footplate work. Ten- or sixteen-power magnification is used at this stage. In approximately 8 out of 10 cases, the major portion of the footplate is clearly visualized within a triangle bounded by the facial nerve, incus, and the posterior crura of the stapes. Since precise work is possible in experienced hands, there need be no fear of complications involving the facial nerve or labyrinth. Every detail can be studied carefully and existing otosclerotic masses noted.

The operating microscope enables one to disprove the theory that fenestration of the footplate is sufficient to routinely improve hearing when mobilization fails. Blind fenestration of the footplate in reality fractures the footplate, leaving either one-third or more of it attached to one intact crura (usually the posterior crus), thus maintaining ossicular continuity. The microscope also enables one to prove that, should both crura be fractured, but still in contact with a mobile footplate, improved hearing will result. Readjustment of fractured parts may be necessary to yield satisfactory hearing, but it can be accomplished because of accurate visualization.

Varied sharp and blunt picks are used around the margins of the footplate, or it can be scored just behind the anterior crus when fixation is complete. These maneuvers, which must be learned through experience, are combined with transincudal pressure. In approximately two out of five successful cases, mobilization of the entire stapes is possible. The remaining three out of five will have a fracture through the anterior crura and the footplate in its forward portion. In the latter type continuity is retained through the intact posterior crus and a section of the footplate.

The microscope allows the surgeon to check footplate mobility since slight pressure with a pick applied to it without touching adjacent parts will move the head of the stapes and the incus.

I had originally reported that it was possible to obtain acceptable hearing improvement in only 33% of cases. Now, the combination of these three operative steps will yield similar results in 75%-80% of cases—a significant improvement.

The primary consideration of the entire surgical procedure is to preserve the continuity of one or both crura with part or all of the footplate. To do this, one must learn the limiting amount of force which can be applied in the first two steps before the stapes is fractured. The feeling of when to proceed to the next step in this technique can be gained only through experience with the surgery. It can be expressed on paper or in words, but its translation into fact comes only with practice.

Transincudal pressure will mobilize many stapes that otherwise would fracture, using Rosen's original technique. This has been shown in surgery performed on the opposite ears of many patients with an equally fixed stapes where the first side had resulted in a fracture. Combining this procedure with the wedging of the footplate has produced the greatest percentage of acceptable results, in my hands.

There is also the advantage of this technique in those cases where a complete exposure of the stapedius tendon is not possible or in an ear where there is little space between the incus and the malleus, which would make the insertion of the Rosen mobilizer difficult.

Improving the percentage of initially good results is what all of us in this work are trying to do at the present time. The use of the transincudal technique combined with wedging of the footplate will enable the surgeon to significantly increase the number of his patients that are rehabilitated through mobilization.

522 West End Ave. (24).

REFERENCES

1. Rosen, S.: Mobilization of the Stapes To Restore Hearing in Otosclerosis, New York J. Med. 53:2650-2653, 1953.

2. Goodhill, V.: Trans-Incudal Stapedolysis for Stapes Mobilization in Otosclerotic Deafness, Laryngoscope 65:693-710, 1955.

3. House, H.: Personal Experience With Stapes Mobilization, A. M. A. Arch. Otolaryng. 65:235-244, 1957.

4. Scheer, A. A.: Restoration of Hearing in Otosclerosis By Transtympanic Mobilization of the Stapes, A. M. A. Arch. Otolaryng. 61:513-534, 1955.

Radical Surgery on the Temporal Bone and Labyrinth Operation for Hearing Improvement

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In recent years chemo- and biotherapy has made such rapid strides that it has more or less eliminated operative treatment of acute mastoiditis and acute petrositis. Medical treatment thus has replaced the operative procedure in the inflammatory and especially suppurative stage, for which formerly surgery was resorted to in order to prevent meningeal involvement.

Today, only those cases which become chronic after medical treatment and which do not respond to this form of treatment are considered for surgery.

There is no difference, anatomically, embryologically, or histologically, between the mastoid cells and the petrous cells, and between the bone tissue of the mastoid portion and that of the petrous pyramid. Likewise, there is no difference between both tissues histopathologically in suppurative inflammation. There is no bacteriological or roentgenological difference between petrositis and mastoiditis. The petrous pyramid, the mastoid portion, and the tympanic cavity, namely, the deep parts and the superficial parts of the temporal bone, are inseparably connected. Petrositis has been erroneously held to bring about paralysis of the nervous abducens or neuralgia of the nervous trigeminus, although anatomically the petrous pyramid does not directly contain these nerves.

Anatomically: Petrous pyramid+mastoid portion=temporal bone.

Histopathologically: Petrositis+mastoiditis=osteomyelitis temporalis.

The investigators up to the present time have held these two expressions as not true, because both the histologically or histopathologically true petrositis and the clinically so-called suppurative inflammation of the petrous apex are thought to be separate entities.

It is similar to the relationship between the histopathological osteomyelitis temporalis and the clinical osteomyelitis temporalis. Both of these diseases together constitute one disease, but up to the present time they have been held to be different diseases. Niho in the *Zeitschrift für Oto-, Rhino- und Laryngologie*, Japan (1942) classified the relationships between otitis media, mastoid cellulitis, mastoiditis, petrous cellulitis, and petrositis in a broad and a narrow sense. The chief purpose of our classification is to correct the wrong idea about petrositis held up to this time, which was restricted by Gradenigo's syndrome. As mentioned above, in mild cases that is otitis media and the mastoiditis is pneumatocellulitis temporalis; in severe cases this disease is osteomyelitis temporalis. We are unable to distinguish between pneumatocellulitis and inflammation of the bone tissue.

TABLE 1.—*Classification According to the Grade of Intensity of the Inflammation*

Osteitis s. osteomyelitis temporalis acuta	Pneumatocellulitis temporalis acuta	1. Mucoendosteitis catarrhalis acuta
		2. Mucoendosteitis purulenta acuta
		3. Pneumatocellulitis purulenta acuta
	Osteitis s. osteomyelitis temporalis acuta	4. Osteitis s. osteomyelitis circumscripta acuta
		5. Osteitis s. osteomyelitis diffusa acuta
		6. Osteitis s. osteomyelitis necroticans progressiva acuta

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TABLE 2.—*Classification According to the Presence of Inflammation in the Temporal Bone*

Osteitis s. osteomyelitis (otitis media in the broadest sense) temporalis	Osteitis s. osteomyelitis temporalis acuta (otitis media in the broad sense)	1. Pneumatocellulitis temporalis acuta (otitis media acuta in the broad sense)	1. Pneumatocellulitis temporalis superficialis acuta (mastoidismus in the broad sense)	(1) Tympanitis simplex (otitis media in the narrow sense)
				(2) Pneumatocellulitis mastoidea (mastoidismus)
				(3) Pneumatocellulitis zygomatica
				(4) Pneumatocellulitis squamosa
				(5) Pneumatocellulitis perituharia
			2. Pneumatocellulitis temporalis profunda acuta (petrosismus)	(1) Pneumatocellulitis perilabyrinthica
				(2) Pneumatocellulitis apicis pyramidis
		2. Osteitis s. osteomyelitis temporalis acuta	1. Osteitis s. osteomyelitis temporalis superficialis acuta (mastoiditis in the broad sense)	(1) Tympanitis (otitis media in the narrow sense)
				(2) Mastoiditis
				(3) Zygomaticitis
				(4) Squamitis
				(5) Osteitis s. osteomyelitis tubae
			2. Osteitis s. osteomyelitis temporalis profunda acuta (petrositis)	(1) Perilabyrinthitis
				(2) Apicitis pyramidis
	Osteitis s. osteomyelitis temporalis chronica (otitis media chronica in the broad sense)	1. Osteitis s. osteomyelitis temporalis chronica	1. Osteitis s. osteomyelitis temporalis superficialis chronica (mastoiditis chronica in the broad sense)	(1) Tympanitis (otitis media in the narrow sense)
				(2) Mastoiditis
				(3) Zygomaticitis
				(4) Squamitis
				(5) Osteitis s. osteomyelitis tubae
			2. Osteitis s. osteomyelitis temporalis profunda chronica (petrositis chronica)	(1) Perilabyrinthitis
				(2) Apicitis pyramidis

Classification according to the grade of intensity of the inflammation is shown in Tables 1 and 2.

The histopathologic changes in specimens obtained during radical mastoidectomy and petrosinectomy—radical operation for osteomyelitis chronica of the temporal bone (Niho)—were examined. These changes are illustrated in Figures 1 to 13.

The entire temporal bone is affected as a whole, but we can find in the specimens a mixture of destruction, degeneration, and repair process of various stages.

Histopathologically so-called otitis media chronica in the broad sense is secondary osteomyelitis or osteitis temporalis chronica. Of course, primarily it is pneumatocellulitis temporalis acuta.

Fig. 1.—Tegmen antri.

Fig. 2.—Zygomatic cells.

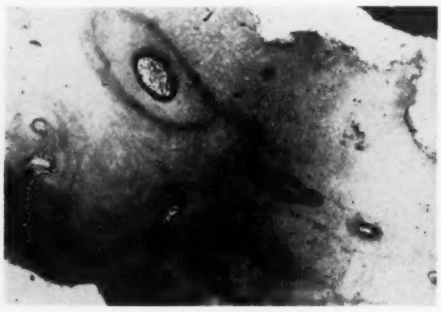

Fig. 3.—Retrosinus cells.

Fig. 4.—Lateral semicircular canal.

Fig. 5.—Around the superior semicircular canal.

Fig. 6.—Posterior semicircular canal.

Fig. 7.—Cell route leading to occipital condyle.

Fig. 8.—Canalis Fallopii.

Fig. 9.—Anterior wall of auditory canal.

Fig. 10.—Ostium tympanicum tubae auditivae.

Fig. 11.—Clinical petrous apex (Niho, Hirano).

Fig. 12.—Petrous apex.

Fig. 13.—Corpus sphenoidale.

The symptomatology of petrositis has until the present time been erroneously held to consist chiefly of paralysis of the n. abducens, and neuralgia of the n. trigeminus, namely Gradenigo's syndrome. Since these nerves have no foramen in the petrous pyramid, their paralysis or neuralgia should be regarded as complications of petrositis. There is a dura layer between these nerves and petrous bone.

Roentgenology has been neglected somewhat in chronic cases. Niho and Takahata have classified the roentgenologic changes in chronic cases as follows: (1) cloudiness; (2) osteomyelitic; (3) bone absorption; (4) sclerotic changes.

Operation for petrositis is as follows: (1) evacuation operation (drainage); (2) direct petrous operation.

In acute cases an important part of the operation is drainage, but in chronic cases it is the fundamental rule in atticomastoid-

Fig. 14.—Healthy temporal bone.

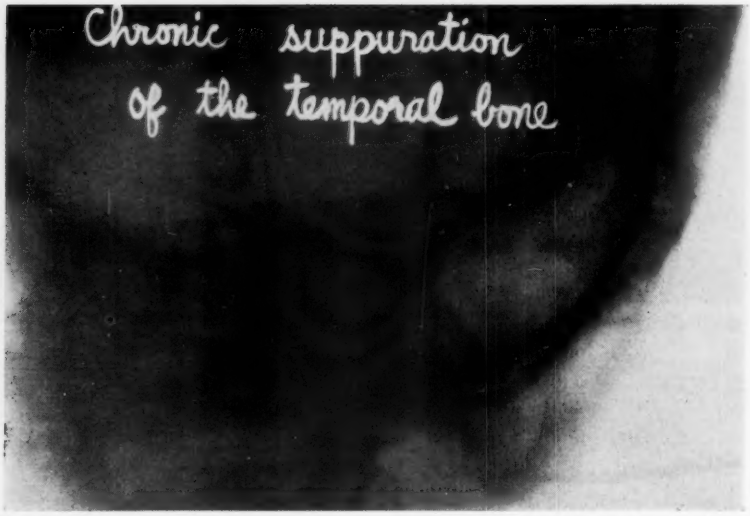

Fig. 15.—This patient had been operated on (mastoidectomy or radical mastoidectomy) three times during three years before visiting our clinic. Because of petrositis chronica the operations were insufficient for complete cure.

ectomy or in radical mastoidectomy to remove the involved bone tissues down to the healthy bone. This rule is also fundamental in the petrous operation. Accordingly the operation is much more difficult in chronic cases than in acute cases. Numerous operative methods have been presented by various authorities, and, practically speaking, it is very seldom that we find only a limited area of inflammation in the petrous bone. Various grades of inflammation can be found in various parts of the petrous pyramid. Therefore, the decision as to the method of operation should depend upon the part involved. It is usually necessary to employ a combination of various operative methods.

In 1942, S. Niho classified these combined operations of the temporal bone as follows: (1) conservative radical operation of osteomyelitis temporalis; (2) radical operation of osteomyelitis temporalis.

Skin incision is performed postauricularly. This incision is more convenient than an endaural approach for the following reasons: (1) elevation of the temporal lobe of the cerebrum and removal of facies anterior to the petrous apex; (2) curettage of posterior, posterior inferior cell routes, and to the cell route leading to the occipital condyle. But I think the skin incision is not so important in comparison with bone disease.

At the present time in Japan the chisel is more convenient than the burr to remove the bony tissue. Involved bony tissue in the deep and fine-structured portion is removed with curette.

We open the antrum first, then remove retrofacial nerve cells, squamous cells, angle cells, peri- and retrosinus cells, mastoid tip cells, and zygomatic cells, etc. Then we curette the involved cells and bony tissue around the semicircular canals, the aditus, and the epitympanum from behind. Special attention is paid to the incus and ligamentum mallei superius. When we find involved cell route or inflammatory changes in anterior superior, posterior superior, and posterior cell routes, curettage is continued into the deep portion until healthy bone is seen.

Next, we enter into the deep portion at the retrofacial nerve cells. The facial nerve is situated in front of this portion; the posterior semicircular canal is above, the

sigmoid sinus behind, and the jugular bulb in the inferior deep portion.

The operative procedures up to this stage do not sacrifice the air-conduction chain, namely the eardrum, malleus, incus, and stapes. The operation up to this stage is called the "conservative radical operation for osteomyelitis temporalis" (Niho, 1942).

When the eardrum is in good condition and the degree of inflammation is slight in the anterior and the anterior inferior cell route, and in the tympanic cavity, it is possible to save the eardrum, the malleus, and the incus, namely, the chain of air conduction, and other portions of the petrous pyramid.

This is a conservative radical operation for osteomyelitis temporalis, as a means of saving the air-conduction chain. The radical operation for osteomyelitis temporalis is the operation involving all of the temporal bone, sacrificing the air-conduction chain.

When the disease is severe and the operation is not adequate, the posterior wall of the auditory canal and bridge are removed; then the soft tissue of the auditory canal is incised. We remove the eardrum, malleus, and incus sometimes before, sometimes after, elevation of the soft tissue of the auditory canal to the outer end of the bony auditory canal.

Next, we chisel the floor of the bony auditory canal until the hypotympanum is visualized. Then we remove the anterior bony wall of the auditory canal, while the patient has his mouth opened, because when the patient opens his mouth, the capitulum mandibulae goes forward and the capsulotympanic space becomes wider. We remove the anterior bony wall of the auditory canal to the bony Eustachian tube. We curette the hypo- and mesotympanal cavity very gently from the peritubal cells to the oval window, especially being very careful to remove granulations with the periosteal layer. After careful curettage of the tympanic cavity, the tensor muscle and tendon are removed; then we enter and curette the petrous cells over the carotid artery. Next, we cut the stapedial crura with granulation tissue, employing stapedial forceps which one of us (S. Niho) devised.

In the majority of acute cases the operation thus far is adequate, but in some chronic cases it is insufficient for complete cure.

The next stage is the removal of the facies anterior of the mastoid portion and the tegmen tympani, utilizing the modified bone-cutting forceps, with wide exposure of the temporal dura. Then the brain elevator which Niho devised is inserted between the dura and facies anterior pyramidis over the impressio trigemini. We elevate the temporal lobe to the petrous apex, and remove the involved bony tissues of the petrous apex, utilizing chisel, curette, and forceps.

We call this operation the "radical operation for osteomyelitis temporalis" (Niho 1942).

In our experience during the past 10 years, both conservative radical operation and radical operation for osteomyelitis temporalis yielded better postoperative hearing, especially in speech perception, as compared with atticomastoidectomy or radical mastoidectomy.

During such operations sometimes we find fistula or necrosis of the semicircular canal. Formerly, we treated such chronic localized labyrinthitis conservatively. For this reason it required a long time before dizziness, headache, aural discharge, etc. were relieved. For about 10 years we have removed necrotic bony tissue and membranous labyrinth down to the healthy labyrinth. Removal of all involved bony or soft tissue, that is, a radical operation for osteomyelitis temporalis, before the labyrinth operation, is necessary to prevent meningitis and postoperative deafness. Dizziness, headache, and aural discharge disappear more quickly after radical operation than conservative treatment.

Usually dizziness disappears several days after partial labyrinthectomy. During the past 10 years we have performed this op-

eration in about 100 cases. In many cases there was an improvement of postoperative hearing.

In 1955 we advocated four grades of operations for the improvement of hearing: (1) conservative radical operation for osteomyelitis temporalis; (2) radical operation for osteomyelitis temporalis; (3) radical operation for osteomyelitis temporalis with fenestration; (4) radical operation for osteomyelitis temporalis with partial labyrinthectomy.

Last year we proposed a classification of the labyrinth operation of the semicircular canal as follows:

1. Removal of the involved bony tissue at the perilabyrinth portion
2. Fenestration operation:
   (*a*) Exposure of the membranous labyrinth
   (*b*) Opening of the exolymphatic space
3. Partial labyrinthectomy:
   (*a*) Removal of granulation tissue in the semicircular canal down to healthy portion
   (*b*) Removal of granulation tissue down to the utriculus
   (*c*) Disappearance of semicircular canal due to chronic inflammation
      (i) Carious bony tissue
      (ii) Compact bony tissue

Involvement of the semicircular canal causes not only dizziness or headache and deafness, but also endangers the life of the victim. When such involved bony and soft tissues from the semicircular canal or the utriculus are removed, the functions of the labyrinth, especially hearing, will return to a certain extent.

Among these operations, for hearing improvement the conservative radical operation in mild cases is the best. But in severe cases, especially for the hard of hearing after radical mastoidectomy, the radical operation for osteomyelitis temporalis (Niho) is the best. In the cases of over 12 months after reoperation the postoperative hearing showed remarkable improvement. They do not have to use a hearing aid. Audiograms of patients who had been operated on (radical mastoidectomy) once, twice, three, four, or more times before undergoing operation for osteomyelitis temporalis at our clinic are shown in Figures 16 to 22.

The purposes of radical operation for osteomyelitis temporalis with labyrinth operation are as follows:

1. Prevention of meningitis
2. Disappearance of dizziness, headache, and aural discharge, etc.
3. Prevention of deafness and improvement of hearing. Postoperative hearing improvement after radical operation for osteomyelitis temporalis and radical operations with labyrinth operation is not so remarkable in audiograms, but the results of a conversational speech-hearing test of 18 cases conducted at a distance of 3.3 m improved to a distance of 5.3 m.

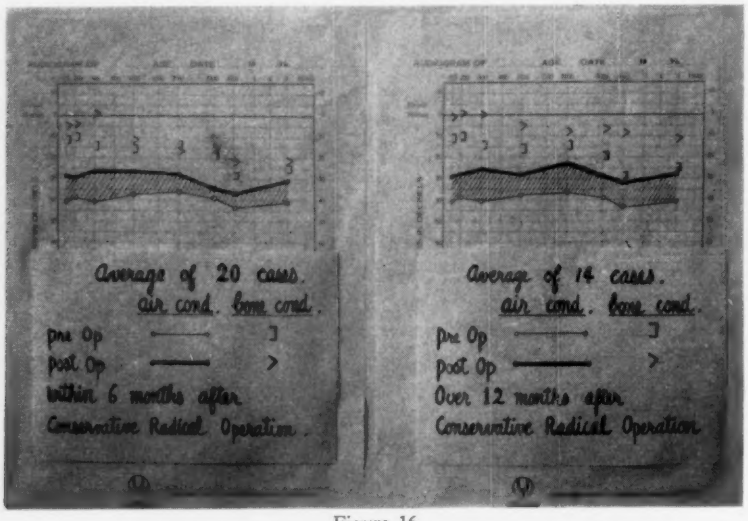

Figure 16

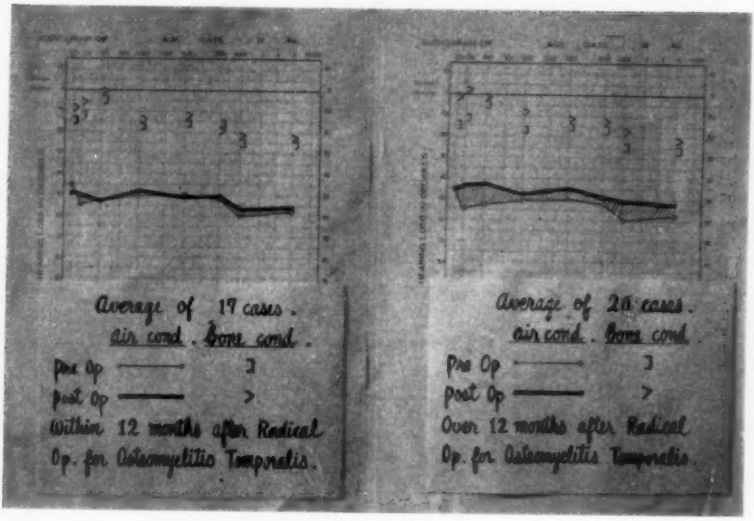

Figure 17

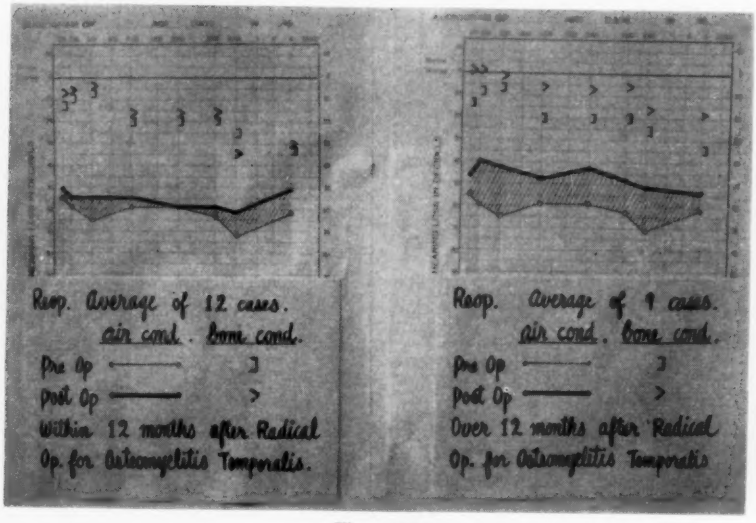

Figure 18

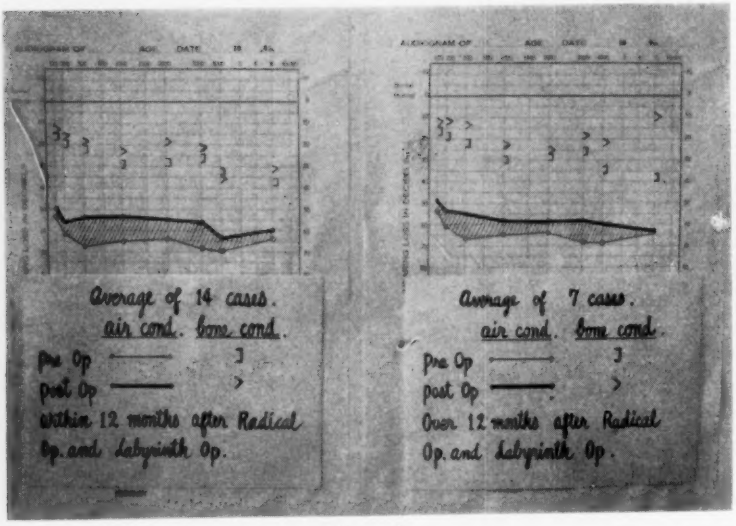
Average of 14 cases.
air cond. bone cond.
pre Op
post Op
within 12 months after Radical Op. and labyrinth Op.
Average of 7 cases.
air cond. bone cond.
pre Op
post Op
Over 12 months after Radical Op. and labyrinth Op.

Figure 19

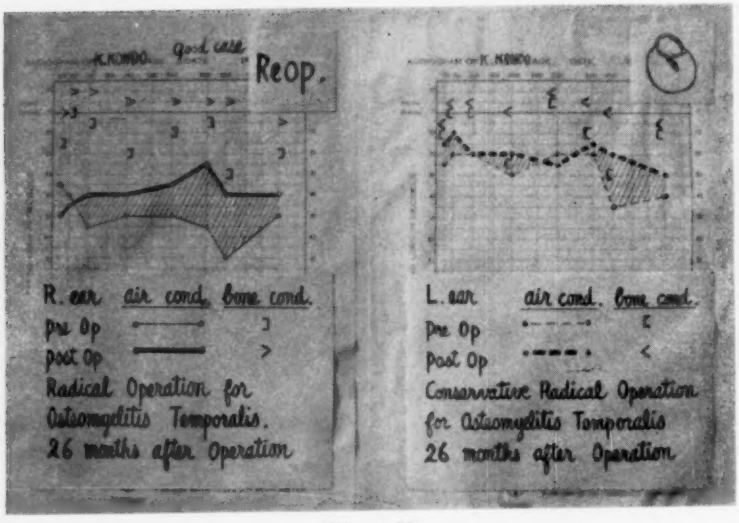

Figure 20

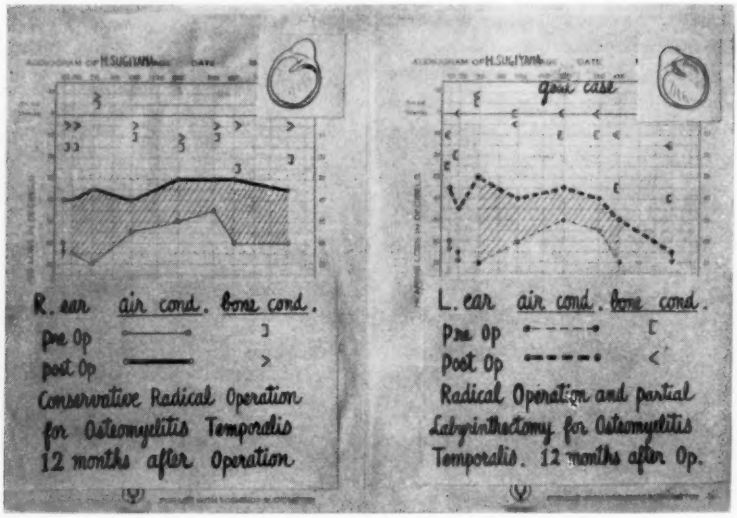

Figure 21

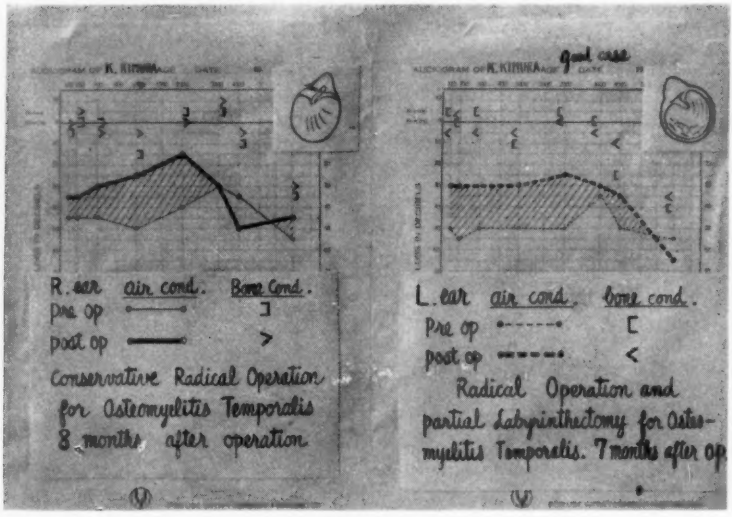

Figure 22

Summary

Niho and his associates in the *Zeitschrift für Oto-, Rhino- und Laryngologie* in Japan (1942) advocated use of the term osteitis or osteomyelitis temporalis (otitis media in the broadest sense) chiefly from a histopathological point of view. Also from a surgical point of view, Niho and his associates advocated conservative radical operation and radical operation for osteomyelitis temporalis (1942).

In 1955, four grades of operation were advocated for hearing improvement:

1. Conservative radical operation for osteomyelitis temporalis
2. Radical operation for osteomyelitis temporalis
3. Radical operation for osteomyelitis temporalis with fenestration
4. Radical operation for osteomyelitis temporalis with partial labyrinthectomy

In 1956, we proposed a classification of the labyrinth operation of the semicircular canal as follows:

1. Removal of the involved bony tissue at the perilabyrinth portion
2. Fenestration operation
3. Partial labyrinthectomy

BIBLIOGRAPHY

Sawatari, J., and Niho, S.: Ztschr. Oto-Rhin-Laryng., Japan, Vol. 48, 1942.

Niho, S.: ibid., Vol. 50, 1946.

Oto-rhin-laryng., Tokyo, Vol. 21, 1949.

ibid., Vol. 24, 1952.

Yokohama M. Bull. 5:125, 1954.

Oto-rhin-laryng., Tokyo, Vol. 27, 1955.

ibid., Vol. 28, 1956.

The Nature of Senile Changes of the Human Olfactory Bulb and Tract

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The senile changes found in the human olfactory bulb and tract are similar to those found in the central nervous system. These changes are characterized by generalized atrophy, loss of neuronal elements, increase of astroglia, and numerous corpora amylacea. In the present study the nature of these changes was investigated in numerous aged persons and in one case which represented "experiment-like" conditions.

Material and Methods

The olfactory bulbs and tracts used for the present investigation were obtained on routine postmortem examinations from persons of age 70 or more. One olfactory bulb used as a comparison with those of aged persons was obtained on autopsy of a 42-year-old woman who 10 months prior to her death had a hypophysectomy because of bony metastases from breast carcinoma. For the removal of the hypophysis a right frontal craniotomy was performed, and an intradural approach to the pituitary was carried out. The frontal lobe was retracted, and the olfactory bulb and nerve identified. A section was made in the proximal portion of the bulb in order to allow further retraction of the frontal lobe. At autopsy the olfactory tract and distal part of the bulb were removed and examined with the rest of the material.

For the histological examination the tissue was partly cut on a freezing microtome and impregnated with variants of the Hortega silver carbonate method.[1] Parts were imbedded in paraffin and stained with hematoxylin and eosin, Masson, Van Gieson, and other methods.

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From the Laboratory of Neuropathology, Neuropsychiatric Institute, University of Michigan Hospital, and the Department of Neurosurgery, Henry Ford Hospital, Detroit.

Description of Material

The examination of the olfactory bulbs and tracts of old persons revealed in the vast majority only moderate loss of neurons and nerve fibers and an increased amount of glial elements, mostly astroglia; numerous corpora amylacea were also present. These changes were found in marked degree in a small group of examined cases, but we were unable to find any correlation between the severity of degenerative changes and the age of the patients. In the latter group the most conspicuous findings were the astrogliosis, which was mostly perivascular, and abundance of corpora amylacea.

Figure 1 shows different stages of transformation of the astrocytes into amyloid bodies. This process represents a degenerative change which begins with the homogenization of the cell body and gradual loss of the processes. In the final stage there remain only round homogeneous darkly impregnated bodies without any resemblance to a cell. Another type of degenerative change in astrocytes is the formation of a ring which surrounds the cell body and apparently is connected to the processes (Fig. 2*A*). Subsequently, the processes break off, and the remnants of the cell body disappear and only the rings are left (Fig. 2*B*, *C*).

The olfactory nerve used for comparison studies was severed from the fila olfactoria and remained with normal blood supply in situ for 10 months. The "experimental value" of this material is reduced by the fact that not only the connection with the first neuron was cut off but also the anterior 2/3 of the bulb was severed, and subse-

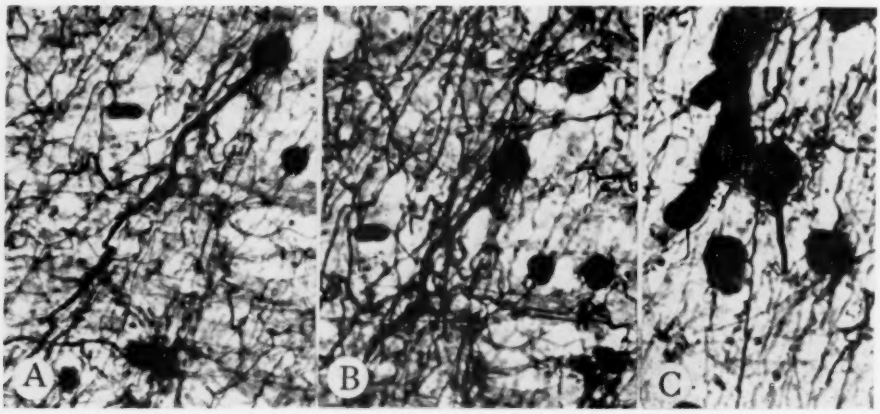


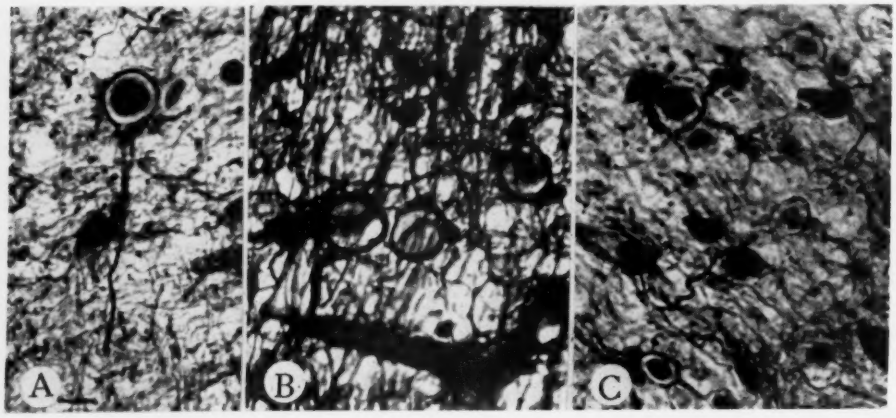
Fig. 1.—Olfactory tract of a senile person. *A*, *B*, and *C* show different stages in transformation of astrocytes into amyloid bodies. Zeiss Neofluar; *A*, *B*, ×40; *C*, ×100.

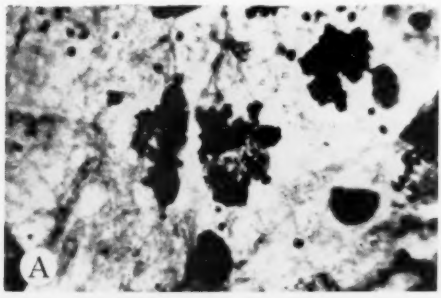
quently the majority of the neurites of the tract lost their ganglion cells.

The histological picture found in this olfactory tract was qualitatively identical with those found in the senile patients, but quantitatively it was much more pronounced.

In the ganglionic layer the large neurons show severe damage, and their bodies are degenerated (Fig. 3*A*); the neurons of smaller variety are also severely damaged, and their processes are swollen and fragmented (Fig. 3*B*). The astrocytes in the proximal portion of the olfactory bulb were markedly increased in number (Fig. 4*A*), and some cells show proliferated processes (Fig. 4*B*). In the olfactory tract are found neurites which are swollen and fragmented (Fig. 5). Figure 6*A* shows the olfactory tract with gliosis and a large amount of perpendicular glial fibers. The central portion of the olfactory tract is filled with corpora amylacea, which are demonstrated in low magnification in Figures 6*B* and *C*. The transformation of astrocytes into corpora amylacea is in some perivascular areas almost complete, as shown in Figure 7*A*. In other areas closer to the ganglionic layer the perivascular astrocytes are still present (Fig. 7*B*).

Fig. 2.—Olfactory tract of a senile person. *A*, (degenerated astrocyte with a ring formation. *B*, *C*, annular formations in progressive stages of degeneration. Zeiss Neofluar; ×100.





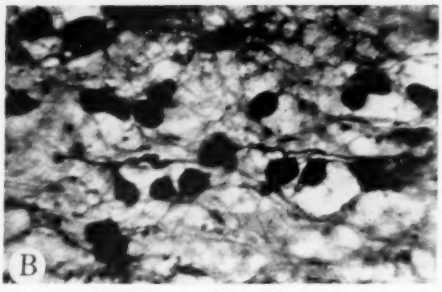
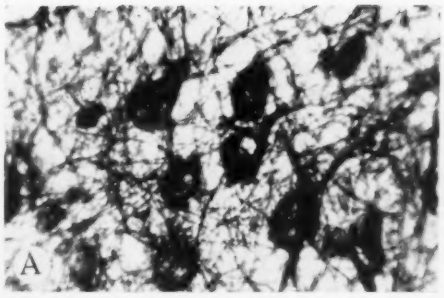


Fig. 3.—Postoperative case; ganglionic layer. *A*, large neurons in progressive stages of degeneration. *B*, small degenerated neuron with swollen processes. Zeiss Neofluar; × 100.



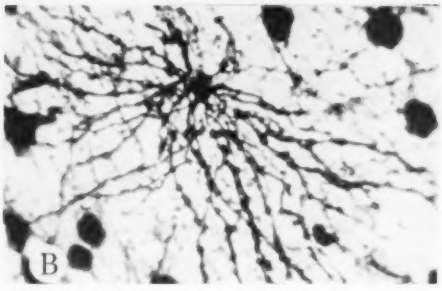
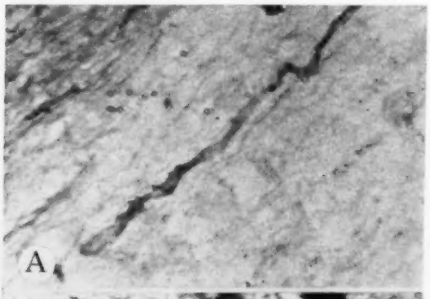


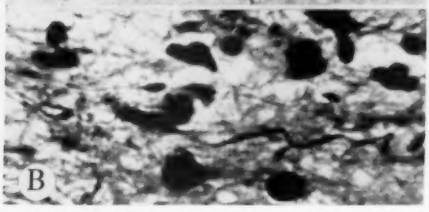
Fig. 4.—Same case as Figure 3; ganglionic layer. *A*, numerous astrocytes, many with degenerative changes. *B*, ganglionic layer close to the layer of the olfactory tract; proliferated astrocyte. Zeiss Neofluar × 100.

Comment

Our present studies were induced by the work of Smith,[2] who defined the ratio of loss of the nerve fibers as 1% a year, beginning at birth. As previously stated,[3] the degeneration of the nerve fibers generally follows a pattern of degeneration similar to that of the senile brain. In the present investigation the severity of the changes could not have been correlated with age. The oldest person examined in the present series was over 100 years old, but the degree of gliosis, amount of amyloid bodies, and the loss of neurons could be classified as mild.

Fig. 5.—Same case as Figure 3; olfactory tract fiber layer. Degenerated nerve fibers with swellings and end bulbs. Zeiss Neofluar × 100.





Since there is a discrepancy between the degree of "senile degeneration" and age, we were interested in determining how much degeneration of the olfactory bulb and tract can be expected in human beings when there is separation of the olfactory bulb from the sensory epithelium of the nasal mucosa. Meurman [4] in his experimental studies on animals has shown extensive degeneration in the olfactory bulb and tract following obliteration of nasal mucosa. Similar although not identical circumstances can be accounted for in a human being in whom a hypophysectomy was performed, and sufficient

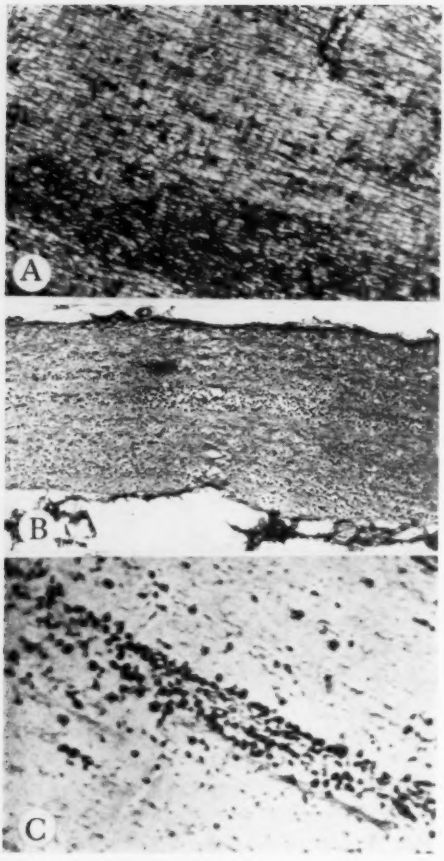


Fig. 6.—Same case as Figure 3; olfactory tract. *A*, numerous glial fibers run perpendicularly to the tract. Numerous amyloid bodies in the central portion. Zeiss 6; ×3. *B*, *C*, amyloid bodies in the central portion of the tract. Zeiss 2; ×5 (*B*); Zeiss 6; ×3 (*C*).

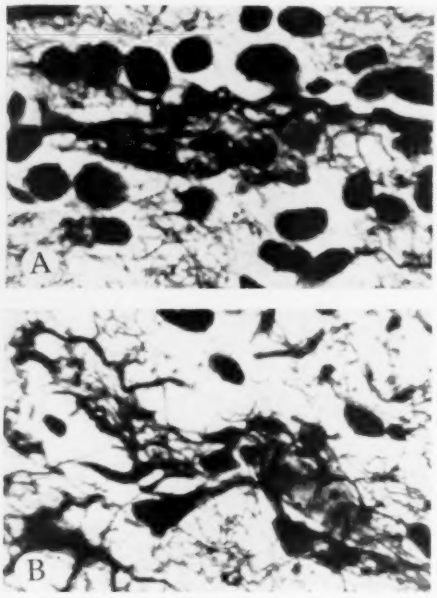


Fig. 7.—Same case as Figure 3. *A*, numerous amyloid bodies in the perivascular space. *B*, perivascular astroglia cells and corpora amylacea in the olfactory bulb. Zeiss Neofluar; ×100.

time elapsed between surgery and death to permit degeneration of the second neuron.

From our findings it can be concluded that in cases where extensive degeneration of nerve cells and fibers is found, the degeneration is caused not by progressive senile processes, affecting the entire central nervous system, but is secondary to degeneration of the first neuron (sensory cells in the nasal mucosa) which is caused by external factors.

The amyloid bodies were previously described in the human optic nerve as end-products of nerve fiber degeneration.[5] In the human retina Wolter[6] described amyloid body formation from either degenerated neurons, nerve fibers, or astrocytes. The present studies confirm the fact that amyloid bodies represent an unspecific end-product of degeneration of astroglia.

In the olfactory tract we were unable to show nerve fibers forming amyloid bodies as end-products of degeneration. However, only few degenerated nerve fibers were present in the postoperative case and none in the olfactories of aged persons. The amyloid bodies were mostly located around the blood vessels and in some areas they replaced the perivascular astroglia almost completely. The slow degeneration of astroglia, secondary to the disappearance of nervous elements which it normally supports, first causes hyalinization of the cell body and later loss of processes.

The glial rings represent another form of astroglial degeneration and are present in aged persons alongside hyaline bodies. In the postoperative case only clusters of the

amyloid bodies were found. These rings were also demonstrated in the brain by general paresis, senile dementia,[7] and Alzheimer's disease.[8]

From the comparison of olfactory bulbs and tracts of senile persons with the postoperative case, it can be concluded that with the degeneration of sensory cells in the nasal mucosa, the second neuron in the olfactory bulb degenerates secondarily. This should explain the severely degenerated olfactory nerves in aged persons.

Summary

The senile changes in the human olfactory bulb and tract correspond to those found in the central nervous system. Extensive degeneration is secondary to destruction of the sensory cells of the nasal mucosa.

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REFERENCES

1. Scharenberg, K., and Zeman, W.: Zur Leistungsefähigkeit und zur Technik der Hortegaschen Silberkarbonatmethoden, Arch. Psychiat. 188:430-439, 1952.

2. Smith, C. G.: Age Incidence of Atrophy of Olfactory Nerves in Man, J. Comp. Neurol. 77: 589-595, 1942.

3. Liss, L.: The Histology of the Human Olfactory Bulb and the Extracerebral Part of the Tract, Ann. Otol. Rhin. & Laryng. 65:680-691, 1956.

4. Meurman, O. H.: Experimental Studies of the Effect of the Pathological Changes in the Nasal Mucous Membrane on the Olfactory Bulb, Acta oto-laryngol. 38:477-483, 1950.

5. Wolter, J. R., and Liss, L.: The Evolution of Hyaline Corpuscles (Cytoid Bodies) in the Human Optic Nerve, Am. J. Ophth. to be published.

6. Wolter, J. R.: Reactions of the Elements of the Retina and Optic Nerve in Common Morbid Entities of the Human Eye, Am. J. Ophth. 42: 10-26, 1956.

7. del Rio Hortega, P.: Alteraciones de las céllulas neuróglicas, Arch. histol. norm. y path. 2:61-63, 1943.

8. Scharenberg, K.: Personal communication to the authors.

Elongated Styloid Process

Symptoms and Treatment

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The symptomatic elongation of a styloid process may explain some instances of pharyngeal and ear pains and some headaches, which have defied exhaustive diagnostic studies. I consider that it is a very neglected otolaryngological entity and that it is very deserving of a niche in the diagnostic armentarium of otolaryngologic practice. I venture to state that most of this group, as medical students, learned something about the styloid process from the anatomy books and the cadaver, but never had the opportunity to put this information into clinical application during the latter part of medical school or during residency training. The otolaryngological textbooks —except for a very few—completely omit any reference to the elongated styloid process, and there has been very little in the literature, even the current journals, to place the proper emphasis on this condition. Radiologists in reporting their findings in skull roentgenograms—seeking for the cause of headaches—fail to describe or include the presence of an elongated styloid process when it is very obvious in the film—yet they will describe a questionable platybasia or enostosis frontalis! The alert dentist recognizes the importance of the elongated styloid process, for he, too, occasionally may encounter such an abnormality, which may impede the performance of his planned work, or he may accidentally fracture an elongated styloid process during his routine procedures.

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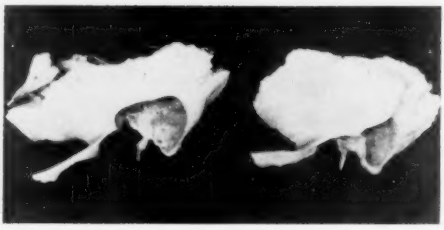

Fig. 1.—Mastoid preparations showing normal length styloid processes.

The normal styloid process is 1 in., or 2.5 cm., in length. The elongated styloid process, which is due to the calcification of the stylohyoid ligament, may be 3 in., or 7.5 cm., or more in length. Three muscles are attached to the styloid process, one extending to the pharynx, one to the tongue, and one to the hyoid bone. There are two ligaments attached to the styloid process, the smaller one to the hyoid bone and the larger one running laterally to the mandible. The distal tip of the normal styloid process lies between the external and the internal carotid arteries. The styloid process originates from the temporal bone on the medial and inferior surface, anterior and medial to the stylomastoid foramen, from which the seventh cranial nerve makes its exit. The process usually points anteriorly and medially, but it may project laterally just inside the mandible; or it rarely may project more posteriorly, being slightly anterior to the cervical spine. The normal-length styloid process is not palpable beneath the tonsillar fossa after tonsillectomy because of the depth of the submucosal tissues. Any process that is palpable beneath the fossa is definitely elongated. Some elongated processes can be palpated through the tonsils. Only about 4% of styloid processes are

found to be elongated, and of these elongations only about 4% are symptomatic. Elongation itself of the process does not necessarily cause symptoms, either with or without tonsillectomy having been done.

The symptomatic elongated styloid process may cause a characteristic and typical pattern of symptoms, and then the diagnosis can usually be made rather easily. However, there are instances where the symptoms are not typical and present a bizarre problem. When searching for the cause of pharyngeal and ear pains and obscure headaches, it is well to bear in mind the possibility of the existence of an elongation of a styloid process. It is very essential, therefore, that the otolaryngologist be familiar with the syndromes produced by such elongations. In previous reports[1-3] of 1937, 1948, and 1949, I have described the two distinct syndromes which could be attributed to the elongation of a styloid process. The first syndrome, which is more commonly found, occurs in patients immediately, or at any time, following tonsillectomy. The other syndrome occurs whether the tonsils have been removed or not. This latter syndrome is found when the carotid artery—either the external or the internal—is impinged upon by a curvature at the tip or in the shaft of an elongated styloid process. These two syndromes will herein be discussed.

Usually the patient relates a rather typical, or certainly suggestive, pattern of symptoms. The usual symptoms caused by the elongated styloid process following tonsillectomy are the following: a sensation of a raw throat, which the patient thinks did not heal properly after operation; a pain referred to the ear, especially noted on swallowing; painful and difficult swallowing; sensation of a foreign body in the pharynx, such as a toothbrush bristle or a fish-bone, and a persistent dull-aching type of sore throat. The pain is due to the stimulation of the sensory endings in the pharynx of the 5th, 7th, 9th, and 10th cranial nerves. Scar tissue, during healing in the tonsillar fossa, tightly binds the mucosa over the tip of the elongated process. Swallowing and talking cause the mucosa to glide, under tension, over the tip of the process. Since no abnormality is visible to the otolaryngologist on pharyngeal inspection, these patients are frequently considered to be neurotics, mainly because they have repeatedly visited the same surgeon with the same complaint; and I can assure you further that they have visited other otolaryngologists also. These patients do have a real pain. The otolaryngologist must be alert.

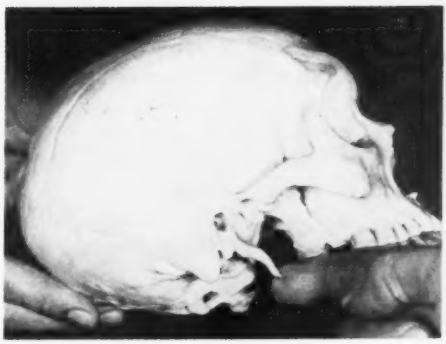

Fig. 2.—Palpation of tip of styloid process beneath fossa.

The diagnosis of the elongated styloid process could almost be made from the typical symptoms given above. However, the actual diagnosis is made by inserting the index finger into the tonsillar fossa and actually palpating the elongated process. Then still further confirmation of the diagnosis is available by roentgenograms. In most instances palpation can be done without causing the patient excessive gagging by avoiding the tongue as much as possible and not allowing the patient to hold his breath during the procedure. If the palpating finger finds a firm resistance beneath the tonsillar fossa, an elongated styloid process is present. If that elongated process is symptomatic, the patient will wince with pain; and all the usual symptoms the patient describes will be accentuated. If the patient has had no symptoms with the elongated styloid process, and it is a casual finding on routine palpation, there will be

no symptoms elicited. No surgery is indicated unless symptoms actually are caused by the elongated process. The elongation of a styloid process may be unilateral or bilateral; and, even then, there may be considerable variation in the amount of the elongation.

The palpation for an elongated styloid process is rather simple and should be a routine part of an otolaryngological examination of the adult—and never omitted when the tonsils have been removed. It is also advisable in adults that tonsils be palpated for an unsuspected underlying elongated process prior to tonsillar operation; for, should an elongated process be found, the surgeon will be prepared to shorten the process during the tonsillectomy. Furthermore, from a legal standpoint, he would have obtained the permission for the shortening procedure at the time of the tonsillectomy. Elongated processes which were not anticipated have caused much trouble to otolaryngologists who use snares during tonsillectomy because of the inability to completely remove the tonsils, with subsequent bleeding episodes frequently associated with tonsillar remnants. It is the elongated styloid process which is not shortened at the time of tonsillectomy that causes the syndrome wherein the patient may have rather marked discomfort following operation. The typical symptoms of the syndrome will persist until the process has been shortened.

Fig. 4.—Elongated styloid process with curvature of shaft.

Fig. 3.—Elongated styloid process with hook at tip.

The second elongated styloid process syndrome involves a carotid artery in which there is carotidynia, with or without a pain throughout the face or head in the pattern of distribution of the external or internal carotid artery, depending upon which artery is impinged upon. These instances are found where the tip of an elongated styloid process is hooked, or where there is marked curvature in the shaft of the styloid process. In either instance there is impingement and tight contact of the elongated styloid process on the carotid artery. Should the external carotid artery be involved, there is facial pain up to the level of the eye. Should the internal carotid artery be involved, there is pain over the entire head from the ophthal-

mic region to the occiput. The pains, referred to by the patient as headaches, are rather consistently nagging, but are not lancinating or knife-like. Palpation of the elongated styloid process in the carotid-artery syndrome, whether the tonsils have been removed or not, will cause accentuation of the pain throughout the distribution of that artery. Some patients actually produce iatrogenic carotidynia by constantly palpating the cervical region over the artery, even when there is no elongation, but the elongated process provides a proper anatomical basis for the syndrome. Undoubtedly, many patients thought to have migraine, histamine cephalgia, cluster headaches, or other neurological disorders have an elongated styloid process impinging on a carotid artery. The elongated styloid process can be ruled out as the cause, simply and easily, by palpation. Furthermore, roentgenograms would be of final assistance. Whether of significance or not, I have noted an excess frequency of elongated styloid processes in hypertensive patients. This finding will bear watching.

In the carotid artery syndrome, just as in the other syndromes, roentgenograms are always made for the confirmation of the diagnosis. In the carotid artery syndrome, however, the posterior-anterior roentgenogram is most valuable in determining the direction in which the shaft of the process points, and it also reveals any curvature of the shaft or the existence of a hook at the tip of the process. The lateral roentgenogram reveals only the length of the styloid process, but it does not give the information relative to the curvature of the shaft or a hook at the tip. Either of these anatomical variations could be in tight contact with a carotid artery. Arterial injection of an opaque substance with roentgenograms reveals clearly the relationship of a styloid process to a carotid artery, but it is extremely rare that arteriograms are necessary, since the diagnosis can be made without them. Ordinary roentgenograms of the skull, both the posterior-anterior and

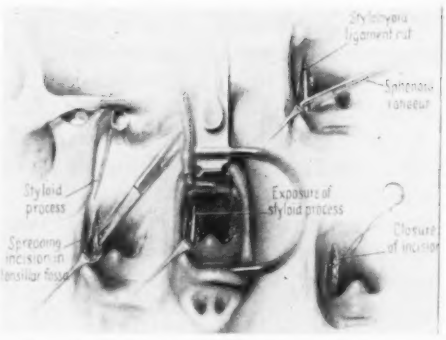

Fig. 5.—Operative field and technique for shortening of elongated styloid process.

lateral, usually give fairly good views of the styloid process which, if inspected with styloid symptomatology in mind, may be of valuable diagnostic service, but special positional roentgenograms for details should be made.

The treatment of the elongated and symptomatic styloid process is the surgical shortening. The fracturing by digital pressure does not relieve the symptoms. A very light general anesthesia of ether administered through the Crowe-Davis mouth gag is used. This gag provides maximum vision by elevating the tongue out of the operative field. Where the tonsils have been removed, a vertical incision is made in the fossa over the palpable styloid tip. By the vertical incision there are very few blood vessels encountered, and frequently the operation is almost avascular. The incision is widened with a clamp or scissors, and then the index finger is used to strip the muscles from the medial and anterior surfaces of the process. The stylohyoid ligament is brought into view and severed with scissors; this then allows the stripping of the remaining muscles and ligaments on the posterior and lateral surfaces of the process with a submucous resection elevator. Ethmoid rongeurs are used to strip up and down the process to remove any remaining muscular or ligamental attachments. After roentgenographic inspection, the desired and calculated length of the process to be resected is removed, after breaking it with heavy ethmoid rongeurs.

The stump is palpated for sharp and ragged edges, which, if present, are smoothed. Care is exercised not to injure the adjacent facial nerve. Sulfanilamide powders are placed in the wound to prevent parapharyngeal space infection. The incision is then closed with plain 0 absorbable surgical sutures. Since the incision is closed, there is much less discomfort to the patient than follows tonsillectomy. There may be some external swelling of the cervical region, which in most cases is due to the sulfonamide drug used. So far, not a single case of parapharyngeal space infection has been observed in more than 150 cases. The patients are able to swallow liquids the day of operation and eat soft food the following day. Hospitalization is usually three days, and the convalescence is usually rapid and uneventful. Invariably all symptoms are relieved by the shortening of the elongated process.

Where the carotid artery syndrome exists and the tonsils have not been removed, it is necessary that tonsillectomy be done first, providing easy access to the elongated process for shortening. In such instances, however, one is not able to close the fossa, and the convalescence is the same to the patient as routinely follows tonsillectomy.

Summary

The symptomatic elongated styloid process may be responsible for two syndromes. The typical and commoner syndrome entails pharyngeal and ear pains following tonsillectomy. The carotid-artery-styloid-process syndrome occurs when the elongated styloid process is in tight contact with the external, or the internal, carotid artery and may cause carotidynia and facial or head pains, whether the tonsils have been removed or not. The intraoral surgical shortening of the elongated portion of the styloid process alleviates the symptoms in both syndromes. The operative procedure has been described.

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REFERENCES

1. Eagle, W. W.: Elongated Styloid Process, Arch. Otolaryng. 25:584 (May) 1937.

2. Eagle, W. W.: Elongated Styloid Process: Further Observations and a New Syndrome, Arch. Otolaryng. 47:630 (May) 1948.

3. Eagle, W. W.: Symptomatic Elongated Styloid Process: Report of 2 Cases of Styloid Process-Carotid Artery Syndrome with Operation, Arch. Otolaryng. 49:490 (May) 1949.

Postoperative Dropping of the Nasal Tip After Rhinoplasty

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Results after rhinoplasty are said to be variable, unpredictable, and uncontrollable; and the portion of the newly fashioned nose most frequently involved is the nasal tip. Secondary dropping of the nasal tip ruins more results in rhinoplasty than any other feature of the operation. The result of rhinoplasty is usually satisfactory at the end of the operation, but four to six weeks later we unfortunately observe that the tip occasionally droops. The nose becomes longer; the cartilaginous dorsum appears too high, and the component parts of the alar cartilages in the tip change in relation to each other. The result is a rounded, unattractive, and unsatisfactory nose.

The causes of secondary dropping of the nasal tip are said to be (1) scar contractures at the septocolumellar suture line, and (2) weight of the lobule. I have tried to stress a third cause: the pull-down action of the depressor septi nasi[2] muscle, which I consider is the most important of the three. I should like to add a fourth—an indirect cause: the failure of the surgeon to lower the cartilaginous dorsum of the nasal septum at operation.

When I think of secondary dropping of the nasal tip, I find it advisable to divide tips into two types: Type 1, the tip with adequate projection; Type 2, the tip with inadequate projection. Type 1 is usually accompanied by a sufficiently long columella, whereas Type 2 has a columella that is too short. The first type is the oversized nose; more frequently operated on than Type 2, and usually more successfully as far as secondary dropping of the nasal tip is concerned. The second type is the snub nose— not so frequently encountered as the other—and it is the "problem child" of rhinoplasty. The reason for differentiating them into Types 1 and 2 is that each type requires a wholly different treatment to prevent secondary dropping. One must differentiate between length and projection of the tip. Length denotes measurement in a caudal direction, whereas projection is the forward measurement directly in front of the face.

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Type 1 Nasal Tip

This is typified by the large oversized nose which possesses a prominent nasal tip with adequate projection and a sufficiently long columella. When secondary dropping of the nasal tip takes place postoperatively in such a case, it is usually due to the fact that the cartilaginous dorsum of the septum was left too high at operation. This, I feel, is the key to the situation and has not been emphasized sufficiently. This mistake in judgment on the part of the surgeon leads to a second mistake in an effort to correct the first one. In order to create a straight profile line and produce the proper esthetic effect, the surgeon is obliged to raise the nasal tip to the level of the too high cartilaginous dorsum of the septum, and this he does by using the so-called orthopedic septocolumellar suture. This suture is applied high on the septum and low on the columella. Just the opposite should have been done; the cartilaginous dorsum should have been lowered to the level of the nasal

tip and approximation sutures utilized between septum and columella. Overcorrection is contraindicated.

When overcorrection is utilized in any rhinoplasty the columella is pulled up unnaturally and placed on a stretch. The depressor septi nasi muscle is elevated out of its natural position in the lip, with the result that the nasal tip tends inevitably to revert back to its normal low position. Secondary dropping results. Overcorrection by means of the orthopedic septocolumellar suture must not be used routinely in rhinoplasty, and certainly not in the oversized nose. When the cartilaginous dorsum is lowered to the proper level and the columella is approximated to the septum in its normal position, no subsequent change in the relative positions of columella and septum will take place and no secondary dropping need be anticipated.

The entire problem can be resolved, provided the cartilaginous dorsum is lowered adequately. But how much is adequately? How do we know when we have lowered the cartilaginous dorsum sufficiently? There is no scientific measuring device or yardstick to guide us. Esthetically, the ideal nasofacial angle is about 35 degrees, depending on the size and features of the individual. This is difficult or impossible to gauge accurately by means of the unaided eye.

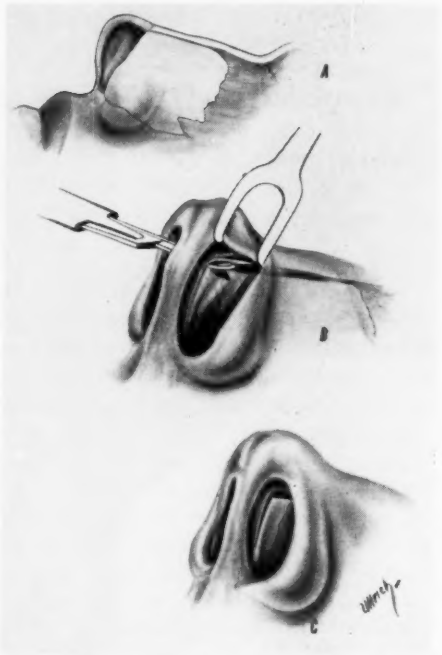


Fig. 1.—Type 1 tip. New technique to determine how far to lower cartilaginous dorsum of septum. *A*, hump has been removed, nose shortened, and cartilaginous dorsum lowered inadequately. Note top of septum and undersurface of alar cartilage at midline are in apposition and nasal tip is resting on septum. *B*, "sliver" of cartilaginous dorsum removed from top of septum. *C*, note 3 mm. space between top of septum and undersurface of alar cartilage at midline, signifying that septum has been lowered adequately. Alar cartilages have not yet been modeled; no septocolumellar sutures have been inserted.

I have utilized a new technique which has helped me in determining the extent to which we should lower the cartilaginous dorsum. The hump is first removed and the cartilaginous dorsum is lowered according to the esthetic judgment of the surgeon. Shortening is then effected by removing a triangular section from the lower edge of the nasal septum. At this point the nasal tip has not been modeled and no sutures have been tied between columella and septum. The rim of one nostril is then lifted ever so gently with a No. 2 ball-point elevator (without raising the tip) in order to inspect the area between the anterior surface of the cartilaginous dorsum of the septum and the undersurface of the nasal tip at midline. The two surfaces should not be in apposition at this time because in that position the weight of the tip could be resting on the septum and the septum could be holding the nasal tip up. What we desire is that the nasal tip should stand up on its own feet, as it were, unsupported by sutures or any artificial means. Under such conditions it will have no tendency to sag. In order to bring this about, a small sliver of the cartilaginous dorsum of the septum should be removed with a No. 15 Bard-Parker blade or scissors, and the two surfaces visualized again. If they are still in apposition, another sliver should be re-

Fig. 2.—Type 1 Nasal tip. Adequate projection of tip, columella of sufficient length, and high cartilaginous dorsum of septum. Corrected by lowering cartilaginous dorsum of septum adequately without raising tip.

moved, and still another until one can observe the slightest interval (2 or 3 mm.) between the two surfaces. When that point has been reached, the cartilaginous dorsum of the septum has been adequately lowered, and the nasal tip is unsupported. It is not artificially propped up but stands up on its own hind legs, so to speak. It will remain so after mere approximation of the columella to septum. The osseous and cartilaginous profile is brought in a straight line with this new level—by filing or other means. After the approximating septocolumellar sutures are tied, the nasal tip can be molded by any technique one wishes to use.

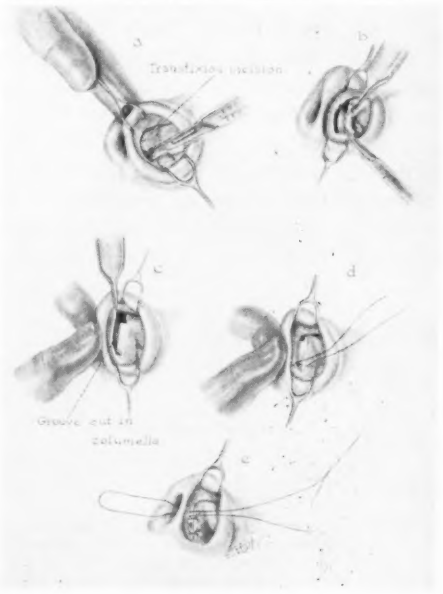
If we overcorrect with the hope that the tip will drop just right, we shall too often meet with disappointment because we are leaving too much to chance. Frequently, it does drop just right; too often it drops too much. Occasionally, it remains too high.

There may be some theoretical objection to the 2-3 mm. supratip subdermal space alluded to on the theory that nature abhors any dead space and will obliterate it by filling it with fibrous tissue. Under such circumstances, an undesirable roundness of the tip could result. This will not occur if the mobile skin over the supratip region and lateral crura (after modeling) are approximated with adhesive tape to the fixed immobile anterior surface of the nasal septum, thus eliminating the space and at the same time creating the desirable slight supratip dip and tilt to the tip.

Another important feature in preventing secondary dropping is the removal of the septal mucous membrane at the lower edge of the nasal septum flush with the cartilage. I realize that a theoretical objection exists that this would result in a rigid or less movable tip but I have not found it objectionable. Superfluous and redundant mucous membrane remaining at the caudal edge of the septum after shortening is followed often by subsequent sagging. When the original transfixion incision is made, the knife should be held close to the lower edge of the septum, and therefore the membranous septum remains attached to the columella. This ensures a movable tip after the septocolumellar sutures are applied.

The intercartilaginous incision should be freely extended laterally as far as the pyriform margin of the maxilla so as to permit

Fig. 3.—Invaginating technique for Type 2 nasal tip. Nasal tip is raised; columella is invaginated onto bare cartilage of caudal edge of nasal septum and held permanently in an elevated position. Septal cartilage extends to but not between medial crura cartilages. (Reproduced from Fred, G. B.: *Ann. Otol. Rhin. & Laryng.* 59:215 (March) 1950, published by Annals Publishing Co., St. Louis.)



liberal rotation of the nasal tip in a cephalic direction after shortening.[3] This maneuver will eliminate buckling and squeezing together of the nasal aponeurosis and nasal tissues in this area. Buckling of the tissues could hinder the upward rotation of the tip and encourage its tendency to return to its former lower position. Free liberal dissection of the external skin of the lateral nasal walls from the upper lateral cartilages and nasal bones is likewise essential.

Type 2 Nasal Tip

This is the snubbed-tip type of nose with inadequate projection associated with a short columella. This is one of the most difficult problems in rhinoplasty, where even the experienced surgeon fails time and time again when conventional techniques are used. This type of tip requires bold, heroic treatment; otherwise secondary dropping of the nasal tip will result.

When the cartilaginous dorsum in this type of nose is high, an attempt should be made to lower it to the level of the nasal tip. This is not always possible. Under no circumstances should the nasofacial angle be made less than 30 degrees. Otherwise, a nose that is too small, and a dorsum that is too low, would result. There is therefore a limitation to how much we can lower the dorsum. Under these circumstances, we are obliged to raise or overcorrect the tip on the septum—the one and only exception to the rule that overcorrection by means of the septocolumellar suture should never be used in rhinoplasty. Overcorrection further poses two problems: (1) how to hold the tip in the elevated position, and (2) how to prevent the depressor septi nasi muscle from pulling it down.

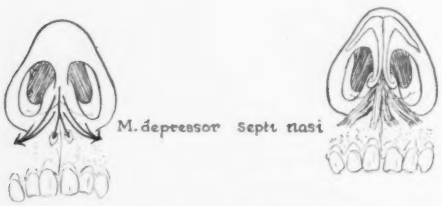


Fig. 4.—Depressor septi nasi muscle, as its name implies, depresses membranous septum, columella, and nasal tip. (Reproduced from Fred, G. B.: A. M. A. Arch Otolaryng. 62:37 (July) 1955.)

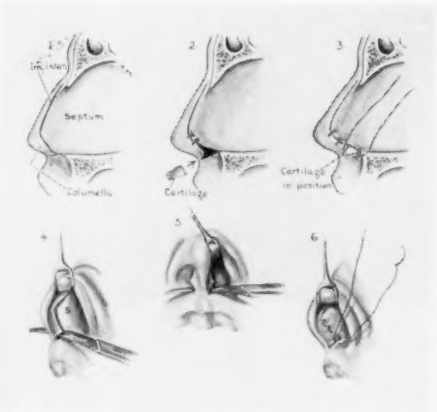


Fig. 5.—Lip-freeing technique. Fibers of depressor septi nasi muscle are cut in columella at a slightly higher level in the columella than is shown in figure. The muscle is thus rendered inactive and ineffectual long enough to result in a firm fibrous adherence between septum and columella. (Reproduced from Fred, G. B.: A. M. A. Arch. Otolaryng. 62:37 (July) 1955.)

The first can be effected by invagination described in a former article.[1] This is a technique which joins the septum and the columella in a manner similar to a carpenter's mortice joint. The caudal edge of the septum is denuded of mucous membrane for 1/8 in. to bare cartilage on both sides, and is fitted into a shallow vertical groove in the cut surface of the columella at the transfixion line. The groove in the columella extends up to but not between the medial crura cartilages, and the columella is therefore not broadened. The skin edges of the columella encircle the denuded caudal edges of the septum. A firm fibrous adherence between septum and columella results over a comparatively wide area, and the tip is permanently held in an elevated position.

The second problem can be eliminated by a technique[2] which cuts across all the fibers of the depressor septi nasi muscle in the columella—thus counteracting and restraining the normal pull-down action of this muscle. This is effected by introducing

Fig. 6.—Type 2 nasal tip. Inadequate nasal tip projection, short columella, high cartilaginous dorsum of septum. Corrected by raising tip with "orthopedic septocolumella suture" combined with invagination and the "lip-freeing" technique.

a No. 15 Bard-Parker blade in the transfixion incision, held horizontally and brought forward at a level just below the lower ends of the medial crura cartilages. A sawing motion of the blade severs both lateral walls of the columella and all the tissue between them up to within 3 mm. of the anterior columellar skin surface. A thin bridge of intact skin still joins the lip to the columella. The muscle is thus rendered inactive and ineffectual long enough to allow firm fibrous adherence to form between septum and columella. Postoperative dropping of the nasal tip is minimized and eliminated to a great extent. The lip can therefore function in its own sphere without any effect on the position of the nasal tip. No claim is made that this technique actually lengthens the short columella. It merely allows us to advance the tip on the columella and hold it firmly in the newly acquired elevated position.

Conclusion

The greatest single mistake in the rhinoplastic correction of the large oversized nose leading to postoperative dropping of the nasal tip is the failure of the surgeon to lower the cartilaginous dorsum of the nasal septum adequately—a mistake which necessitates overcorrection of the tip on the septum. This inevitably results in a return of the tip to its original low position. Overcorrection is contraindicated in this type of tip. A new technique is described which determines and pinpoints with exactness when the dorsum has been lowered adequately.

The snubbed type of nasal tip characterized by inadequate forward projection and short columella results almost routinely in failure when conventional methods of correction are used. Overcorrection of the nasal tip on the septum by means of the orthopedic septocolumellar suture, invagination, and elimination of the pull-down action of the depressor septi nasi muscle are mandatory and afford the best chance of minimizing secondary dropping of the nasal tip characterized by inadequate projection.

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REFERENCES

1. Fred, G. B.: Nasal Tip In Rhinoplasty, Ann. Otol. Rhin. & Laryng. 59:215 (March) 1950.

2. Fred, G. B.: Role of Depressor Septi Nasi Muscle in Rhinoplasty, A. M. A. Arch. Otolaryng. 62:37 (July) 1955.

3. Gilbert, J. G., and Feit, L. J.: The Nasal Aponeurosis and Its Role in Rhinoplasty, A. M. A. Arch. Otolaryng. 61:433 (April) 1955.

Rehabilitation of Patients Following Glossectomy

Attempts to Reestablish Articulation and Maintain Normal Nutrition

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From earliest times, the tongue has been associated in the public mind with the production of speech. Excision of the tongue was the penalty often selected as suitable punishment for those committing crimes of heresy or blasphemy. This mutilation was intended to prevent any further utterance of an antireligious nature. It was noted, however, that many victims of this ancient punitive measure retained the ability to speak, even when the tongues were cut out "as low down as the throat." [1] This supposedly miraculous return of the power of speech was attributed to divine intervention.

Excision of the tongue in modern times is generally limited to cases of malignancy or trauma. Although popular opinion is still of the belief that one's tongue is necessary in order to speak, the almost total lack of reference to rehabilitation of glossectomy cases in the literature might imply no problem exists. It has been stated that cancer of the tongue is the most frequent location of malignancy in the head and neck, and that this comprises 2% to 3% of all human cancer.[2] Further studies indicate that cancer of the floor of the mouth and tongue develops each year in approximately 200 patients per 1,000,000 population in the United States.[3]

Since the function of the tongue is a much more basic and biological one than speech, the factors of chewing, sucking, and swallowing are impaired as a result of glossectomy. Early management and control of the patient after surgery will hasten the restoration of normal deglutition and mastication with use of remaining structures as substitute mechanisms.

Two glossectomy cases and an outline of specific therapy employed will be described in this paper.

The chief complaints of the patient in both instances were continuous and uncontrolled salivation, inability to swallow solid foods, regurgitation of liquids through the nose, restriction or absence of movement of the lips, jaw, and neck, and unintelligible speech.

CASE 1.—A 61-year-old white man with extensive carcinoma involving the lateral, ventral, and dorsal surfaces of the tongue, floor of the mouth on the right side, plus enlarged nodes in the right side of the neck. A right radical neck dissection of the composite type was performed—including the right sternocleidomastoid muscle, all areolar tissue from the posterior cervical triangle, the internal jugular vein, the tip of the parotid, the submaxillary gland, the right half of the mandible, and approximately three-fourths of the tongue.

Speech therapy was initiated 20 days after surgery. Upon initial evaluation of the patient it was noted that he was unable to open his mouth more than 19 mm. A constant flow of saliva seeped through this small aperture. Cervical neck movements were severely restricted. The patient's attempts at speech were muffled—voice quality was extremely nasal and hoarse. Articulation was largerly unintelligible and characterized by omissions, substitutions, and distortions. Nourishment was administered through a Levin tube.

CASE 2.—A 68-year-old white man with a history of epidermoid carcinoma, high-grade malignancy of the right lingual tonsil, and keratonizing squamous-cell epithelioma of the left floor of the mouth, which had been treated with irradiation. Six months later, examination revealed residual carcinoma in the left anterior floor of the mouth requiring a partial glossectomy, removal of the floor of the mouth, resection of a portion of the left mandible, and a left radical neck dissection.

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The patient requested speech therapy 25 months after surgery, with complaints of salivation, difficulty in swallowing, and unintelligible speech. Initial examination revealed the absence of the left mandible, which caused the lower lip to retract and approximate the alveolar ridge. The mouth aperture was 32 mm. when fully opened, but, because the lips did not meet, an opening of 17 mm. existed even when the mouth was closed. Saliva flowed through this space. The neck was held in extension; movement in any direction was absent. In the two years since surgery he had existed solely on fluids, which were swallowed with considerable difficulty. Much of the liquid was regurgitated through the nose.

Therapy for these two cases was essentially identical. Diphenhydramine (Benadryl) 50 mg. t. i. d. to control sialosis was prescribed. Stretching, massage and resistive exercises to the following muscles were administered to increase the mouth aperture and decrease contractures of the cheeks and lips: mentalis; risorius; zygomaticus; caninus; quadratus labii superioris and inferioris; orbicularis oris; buccinator; platysma; pterygoideus internus and externus; temporalis; masseter; digastric; suprahyoid.

Stretching activities to increase the range of circumduction of the neck consisted of passively flexing, then rotating, then extending the neck in a slow rhythmical pace, approximating the chin with the glenohumeral joints in the lateral positions.

Feeding was achieved by first using a small plastic pastry tube of the plunger type which was filled with diluted infants' food and inserted well into the oral cavity. Small quantities of food were introduced into the faucal region by pressing the plunger. The patients were instructed to "hold their breath" and swallow. By consciously elevating the soft palate, the nasal passage was closed off, and swallowing was performed without regurgitation. As mobility increased in the lips and neck, long-handled spoons were substituted for the pastry tube. At the cessation of therapy, the patients were able to eat such foods as diluted ground beef and purée soups, using normal implements.

The patients were seen daily for half-hour sessions over a period of four to five months. It should be emphasized that therapy consisted primarily of increasing the facilities of chewing, sucking, and swallowing. As these skills improved, and salivation was controlled, a definite change was witnessed in the articulation and intelligibility of speech.

At the time of discharge, the only sounds consistently absent were (k), (g), and (ng). Tongue-tip consonants, such as (t), (d), (l), (n), (s), and (z) were most often present in the initial position, and less frequently heard in the medial or final positions. The vowels were all nasalized. In Case 2, some of the tongue-tip sounds were produced by pressing the lower lip against the alveolar ridge. Normal range of motion of the cervical neck was achieved by both patients.

The actual therapeutic techniques employed were approved and supervised by the Medical Director of the Speech Department.

Summary and Conclusion

Two cases of glossectomy and radical neck dissection have been discussed regarding therapeutic techniques employed to improve chewing, sucking, and swallowing.

It is possible with training to establish a substitute form of articulation in a glossectomized patient resulting in intelligible speech.

The success of the training appears to depend upon (*a*) extent of mobilization and return of flexibility of muscles of facial expression, mastication, and muscles of the neck, (*b*) control of salivary secretion.

Normal swallowing and feeding activities can be induced by the use of devices designed to project food into the faucal region.

St. Vincent's Hospital.

REFERENCES

1. Martin, H. E.: The History of Lingual Cancer, Am. J. Surg. 48:703-716, 1940.

2. Martin, H. E.; Munster, H., and Sugarbaker, E. L.: Cancer of the Tongue, Arch. Surg. 41:888, 1940.

3. Hendrick, J. W., and Ward, G. E.: Treatment of Intraoral Cancer, J. A. M. A. 150:1099-1103, 1952.

An Explanation of the Pathogenesis, Physiology, and Therapy of Meniere's Disease

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The search for the singularly most efficient and permanent while least debilitating therapeutic approach in the treatment of Ménière's disease, or hydrops labyrinthi, has concerned investigators ever since Hallpike and Cairns published their findings on this disease in 1938.

The range of therapies runs the gamut from the nonsurgical managements by diet control (Furstenberg, Lashmet, and Lathrop) and vitamin dosage (Selfridge), over the streptomycin (Hanson), nicotinic acid (Atkinson), and histamine (Derlacki, Sheldon and Horton, Shambaugh, Lillie et al.) routes, down the unmarked trails of infected foci (Wright, Kemler), through the intersection of eighth cranial nerve surgery (Crowe, McKenzie, Green and Douglass), along the byways of stellate ganglion injections or dorsal sympathectomy (Passe, Johnson, Strong) and finally, home to the complete or partial obliteration of the labyrinthine apparatus (Cawthorne, Day, Goodyear, Perlman, Portmann).

Conflicting Etiological Beliefs

The wide variations in these therapies can best be attributed to the variations found in the beliefs concerning the etiology and pathogenesis of labyrinthine hydrops as advanced by writers on the subject. Examination shows them to be rather indefinite and at times more than a little conflicting.

Thus Lindsay considers hydrops to be the result of autonomic dysfunction which causes a disturbance of the secretory apparatus leading to a hypersecretion of endolymph and a dilation, or hydrops, of the endolymphatic spaces due to the osmotic pressure changes which occur.

Hilger also considers the pathogenesis a result of autonomic dysfunction which effects angiospastic change in the labyrinthine arteries and thus labyrinthine ischemia.

Williams says the disease is the result of allergic reaction (which he feels is synonymous with autonomic dysfunction) affecting the endolymphatic vascular system due to a spastic-atonic state producing a localized extracellular edema.

Conversely, Cawthorne and Hewlett in their analyses of over 900 case histories find little to support allergy as a significant etiological factor since less than 5% of the cases studied had allergic symptoms. Moreover, the average age of onset in endolymphatic hydrops (44 years) appears to differ significantly from that of the average onset age of allergy (under 40 in 80% of the cases).

Gundrum, on the other hand, in his evaluation of 100 cases of Ménière's disease supports allergy as an etiologic factor but lists infections and trauma as deserving some consideration.

In 1952 Lempert, Wolff et al. pointed out the inconsistencies in the hypothesis of hydrops pressure as the triggering mechanism of Ménière's syndrome and presented evidence that suggests endolymphatic hydrops to be a chronic progressive herpetic neuritis either of toxic or trophic origin.

Fowler, meanwhile, emphasizes abnormal blood physiology as the important factor in the etiology of Ménière's disease.

Derlacki, subscribing to the "multiprecipitator" doctrine affecting the autonomic system, reports a "definite finding

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of a specific antigen antibody reaction in his "most completely unrewarding search 5-10% of the cases." Derlacki reports that (was) for foci of infection."

Into this last statement Wright fires a barrage of impressive statistics on his results in 88 cases of Ménière's disease treated by removal of focal infections. His surgical removal of dental, nasal, and pharyngeal sepsis resulted in marked improvement in over 80% of his cases in one group studied and followed for an average of more than three years. This proportion surpasses by considerable margin those results reported with utilization of other therapeutic means. Furthermore, the length of time covered in Wright's follow-ups (1-10 years) indicates more than temporary relief from symptoms, a result which other therapies find hard to duplicate (Strong, Derlacki, Hanson).

Goodyear, in outlining his labyrinthotomy technique, mentions the necessity of first ferreting out possible foci of infection before resorting to the obliteration of the labyrinth.

Recently, Kemler presented a summary of the leading therapies now practiced with regard to Ménière's disease, giving his own previously published inclinations as to therapy—that of eradication of focal infection—some discussion. Kemler, emphatic in his contention that focal infection lies at the etiological basis in Ménière's disease, cites Harley's two principal objections to the focal infection approach in the management of hydrops labyrinthi: "(1) The failure to achieve good results by surgical means in the majority of cases in which a localized focus, capable of removal, was found; and (2) The failure to find a focus of infection at all in many cases."

Focal Infection Rationale

Over a period of years we have subscribed to and utilized the focal infection approach in the treatment of labyrinthine hydrops with excellent results. We support Kemler's contention to Harley's two criticisms of focal infection treatment, viz., "that there may be more than one focus of infection which is overlooked or overshadowed by the more obvious one; and that there are areas of infected foci which are not considered as such by others."

But even as our results have been highly successful and extremely gratifying in terms of permanent remission from symptoms of vertigo, nausea, and vomiting, and a reduction of tinnitus, together with sometimes even a partial (minimal) recovery of hearing loss (Fig. 1), our animal research and attempts to prove the physiological mechanism and/or pathogenesis of hydrops which could indisputably account for this marked degree of success has been quite unsuccessful. One can be confronted with the almost unbeliveable recovery in his patients, and one can hypothesize as to the mechanisms and relationships involved in these recoveries, but these hypotheses must be confirmed or denied in the harsh light of factual physiological proof and/or on an anatomicopathological basis. For the focal infection theory this proof now appears evident. On the basis of Young's work, the focal infection advocates have now acquired anatomical and physiological evidence to support their contentions.

Anatomist Proves Lymphatic Channels

Young studied the temporal bone and associated regions by means of metal casts, roentgenograms, and dyes. He found lymphatic channels proceeding from the labyrinth to the middle ear, thence along the Eustachian tube to the retronasopharyngeal lymph nodes. The petrotympanic (Glasserian) fissure enters the mandibular fossa as an outlet from the middle ear and transmits the chorda tympani which contains lymph channels from the petrous bone. The tympanic bone, with similar lymph channels, forms the posteromedial boundary of the mandibular fossa adjacent to the tympanum and Eustachian tube.

The discovery of this direct anatomical relationship between the labyrinth and the mandibulopharyngeal apparatus in the

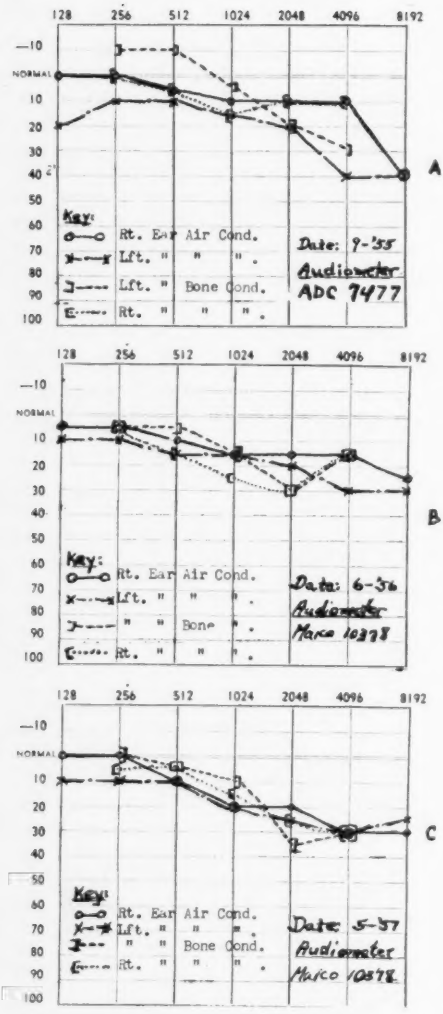


Fig. 1.—Audiometric configurations on a 33-year-old housewife afflicted with Ménière's disease. Patient reported for initial audiometric evaluation in weakened physical state, complaining of head fullness, marked left ear tinnitus, malaise, nausea, and attacks of extreme vertigo. She was unable to walk unassisted. Symptoms had been present approximately one month.

Audiogram *A* shows left ear bone conduction elevated on low and mid frequencies and air conduction loss at 4096 and 8192 cps. Right ear configuration within normal limits except for 40 db. loss at 8192.

Audiogram *B*, plotted nine months after initial audiogram and alveolectomy, shows left ear air-conduction gains at 4096 and 8192 cps of 10 db. with bone-conduction curve coinciding more closely with air-conduction curve. Gain of 10 db., left ear, at 125 cps is perhaps an audiometer artifact. Right ear curve is essentially the same as Audiogram *A* except for 15 db. gain at 8192 cps.

At this testing patient reported complete cessation of vertigo, nausea, and head fullness with no recurrences since surgery. A marked improvement in physical condition was evident. She walked unassisted and had resumed all household duties. Tinnitus, though noticeably reduced, was still present and annoying. She reported that quick head changes (from lying to sitting position) at times produced momentary lightheadedness.

Audiogram *C*, plotted one and a half years after alveolectomy, at which time patient was recovering from a head cold. She reported no recurrence of Ménière's symptoms, was enjoying good health, and expecting another child in five months, with her pregnancy to date termed as "easy and pleasant."

Audiometric configurations indicate that hearing had apparently stabilized at level of Audiogram *B*.

Rhesus monkey gives rational and etiological insight to Wright's findings. In one group of 30 cases studied who had some idea as to their individual precipitating cause, Wright reports that 15 (50%) reported onset of symptoms coincidental with dental extractions, while tonsillitis, tonsillectomy, influenza, and pneumonia accounted for 6 (20%) of the cases. The remaining nine cases (30%) gave trauma, surgical interventions, and childbirth as precipitating causes.

Considering Young's anatomical findings and Wright's statistics, Hoople's suggestion that focal infection may be one of the causes of changes in blood supply and fluid balance in the ear appears to be an understatement. More emphatically, focal infection appears to be *the* important cause of blood and fluid balance changes in the ear, especially in cases of labyrinthine hydrops. We will support this viewpoint in our discussion of the physiology of this mechanism later.

Diagnosing Infected Foci

In our search for the etiological basis of Ménière's disease we found that focal infection played a predominant role and was

found in every case observed by us. It is our opinion that other diagnosticians have overlooked or failed in their search for a focus of infection because they look primarily for pus. This despite the fact that medical science is aware of numerous diseases of known infectious origins which do not manifest pus formation. Syphilis and typhoid fever are examples of such. Tuberculosis (except, perhaps, in its acute form as in caseating bronchopenumonia) is another.

In patients afflicted with hydrops labyrinthi, besides the usual physical and audiological symptoms, almost without exception we found dental disease, sinus disease, or both, even though pus does not commonly appear in and about infected teeth. Furthermore, two outstanding physical findings were apparent in our series of cases, one or both of which were always present.

Alveolar Sensitivity

First, if the examiner passed his finger with constant pressure beginning at the midline of the upper alveolar process and proceeding posteriorly to the area of the first molar tooth, the patient often complained of sensitivity on one or both sides. As the examiner proceeded back to the tuberosity with continued pressure, the patient would almost without exception complain of rather severe tenderness. On occasion the mere examination of the patient in this manner precipitated rather severe vertigo, and in three of our cases a typical vertiginous and tinnitus attack was produced by this manipulation. Not infrequently, this sensitivity to pressure stimulation extended to the anterior border of the ramus of the mandible. In addition, if the examiner placed a tongue depressor or two between the molar teeth on the sensitive side and requested the patient to bite down vigorously, symptoms of Ménière's disease would often be precipitated. (We have taken motion pictures of this phenomenon.)

Mandible-Maxilla Space Variations

Secondly, while we were engaged in the above procedure, it was noticed in a number of instances that the distance between the ramus of the mandible and the maxilla on one side differed considerably from the equivalent distance of the structures on the other side. Usually the affected side showed less space between the ramus of the mandible and maxilla than did the unaffected side. In these instances reactions to the tongue-depressor bite test resulted in (1) a decided increase in symptoms of tinnitus and vertigo, or (2) a noticeable reduction in them. In our opinion, when the above findings presented themselves, it became obligatory for the examiner to search for an etiological basis.

Findings in the first category suggested the almost self-evident diagnosis of dental-alveolar pathology of some sort, highly suspect of infection. Therefore we resorted to dental x-rays for further diagnosis and confirmation.

Findings in the second category, in our experience, were always present in patients with overbite or so-called "closed bite", or in cases of protrusion due to malocclusion. This suggested that a mechanical impingement of the lymphatics could be capable of impairing the lymphatic drainage mechanism of the labyrinth. This difficulty is rather simple to overcome if the laryngologist has the cooperation of a competent dentist. Thompson outlines an approach to balancing and opening the bite that gives excellent results, and whether or not Costen's explanation for this finding pertaining to the temperomandibular joint is correct, or whether the explanation of Weinmann and Sicher is right, appears irrelevant in so far as correction of the pathology is concerned.

Routine Diagnostic Sinus X-Rays Inadequate

Our searches for the aforementioned oral infections invariably led to and centered in the sinuses and in the teeth. In the diag-

Fig. 2.—X-ray plate of posterior upper teeth showing thickened antral mucous membrane, pulp stones, stripping back of periodontal membranes with infected pockets, teeth protruding into antral floor, and inflammation of soft tissues. (Radiologist's diagnosis of Water's view was negative in this case.)

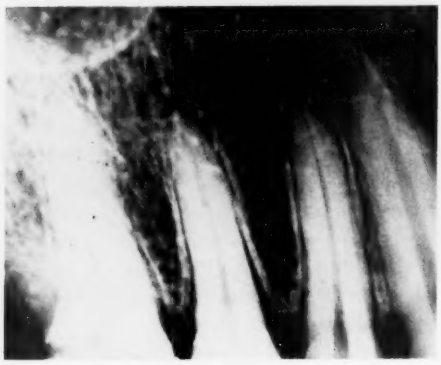


Fig. 3.—X-ray plate of upper posterior teeth showing loss of bone trabeculation, thickening and stripping back of periodontol membranes, and thickening of mucous membrane of the antrum.

nosis of sinus disease we found that the ordinary x-rays of the paranasal sinuses were not adequate. However, we found that a most efficient and accurate way to see thickening of the membrane of the antrum was to take intraoral dental films with a "soft" technique. Such films readily revealed the thickened membrane present at the antral floor even after diagnostic sinus irrigation yielded nothing (Figs. 2, 3). Many times x-rays of the paranasal sinuses, even though the Water's view was taken well, did not show this change which became evident on the dental x-rays.

Diagnosing Dental Disease

In the search for dental disease one must keep in mind the almost axiomatic statement that if a structure is not normal, then it it abnormal; and if the abnormality is the result of disease, then it is pathological. Since most, if not all, dental changes are the result of disease we must consider such findings as pathological. Thus a tooth whose crown consists of some dentine and some amalgum is not normal and one must take issue with the dentist who considers it as "normal." No tooth, indeed no organ, whose normal structures are replaced by artifacts can be called "normal." Moreover, such dental changes which manifest

Fig. 4.—X-ray plate of thickened mucous membrane in floor of antrum with mucocele adjacent to the root of the tooth extending into the antral floor; dental pulp stones and deep intradental pockets are evident.

Fig. 5.—X-ray plate of alveolar processes seven years after teeth extraction for pyorrhea. Shadings indicate abscess formation, loss of trabeculation, and general residual infection in alveolar bone.

Fig. 6.—X-ray plate of upper posterior teeth showing retained tooth root with abscess. Abscess within antrum at the root tip is evident, and dental pulp degeneration with stone formation together with intradental pockets can be seen.

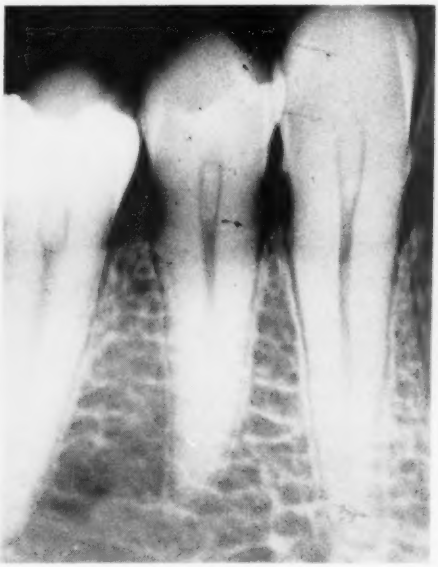
Fig. 7.—X-ray plate of lower teeth showing hypercementosa, root-tip clubbing, loss of trabeculation, and thickening of periodontal membrane.

themselves as pulp stones (Figs. 2, 4, and 9), thickening or stripping back of the periodontal membranes (Figs. 2, 3, 7), crowns, filling, and root canal resections (Figs. 6, 7, 9), and of course, any hyperostosis (Fig. 7), any rarefication or loss of trabeculation of the bony structure surrounding the teeth (Figs. 3, 5), any inflammatory reaction in the soft tissues, and any resorption of bone around the teeth (Figs. 2, 3, 5, 7, 8) must be considered abnormal, pathological, and therefore potentially hazardous to the patient's well-being.

Another common pitfall in overlooking dental infection is in the case of the edentulous patient. We have found that though the patient be edentulous, one cannot overlook the possibility that he might well, and in fact usually does, have disease of the dental arch (Fig. 5). Why the dentist thinks he has removed the infected foci if it is due to a periodontoclasia or pyorrhea by simply

Fig. 8.—X-ray plate showing alveolar bone destruction in a case of far advanced pyorrhea.

Fig. 9.—X-ray plate showing dental pulp stones and pulp degeneration.



extracting the tooth or teeth is difficult for one to comprehend. (See Blum: "Facts vs. Opinions," for a discussion of this fallacy.) It is not adequate to extract a tooth for pyorrhea (Fig. 8). Rather it is imperative that all the diseased bone be removed in order to eradicate a focus of infection. Blum, Hargrove, Reading et al., and others discuss and present statistics indicating that infection of the posterior upper teeth often extends to the antral floor (Figs. 2, 6). When such is the case, mere dental extractions are less than useless and the routine Caldwell-Luc procedures are, of course, rendered rather innocuous. It becomes necessary in such cases for the oral surgeon and the otolaryngologist to work together simultaneously on the patient.

Removing Dental Infections

Previously, Murray discussed and described in detail the methods and techniques of alveolectomy used in eradicating retained dental infections. The technique included, as well as the extraction of teeth, the surgical removal of the bony septa between the teeth, and the buccal, labial, and lingual plates of both jaws. All infected bone is removed until a level of good blood supply is reached. No septi should be left as ivory-like structures but should be cut back until bleeding is punctate. The wounds are sutured and irrigated by hypertonic saline solution, and healing by granulation, not per primam, is always the goal. Myers and Sylvester subsequently cited this method as being most effective in the removal of persisting dental pathology.

As we have never had any cases that cleared up from the ordinary Caldwell-Luc procedures alone, we feel that in most cases the infection is more deeply seated in the bony structures in and about the teeth. Interestingly enough, our series of cases did not include a patient with infection around the Eustachian tube per se, or of the dorsum of the tongue as Kemler mentions; however the possibility of such infections existing either as a primary focus or as a secondary focus cannot be denied. On the other hand, we have found that after removal of the dental and sinus infections, the infections present in the nasopharynx very often drain spontaneously and disappear.

Another point worth mentioning, as far as our cases were concerned, is that we did not see a case of true Ménière's disease cleared up by merely balancing and opening the bite. We have, however, seen cases of head and neck pain accompanied by dizziness which resemble Lindsay's descriptions of so-called "pseudoMénière's disease" and therefore might be classified as such. These cases were helped considerably by opening the bite and balancing the occlusal planes.

Succinctly then, two things must be made perfectly clear as far as the focal infection treatment of Ménière's disease is concerned: 1. The complete eradication of infected foci includes more than the mere pulling of teeth, or the routine Caldwell-Luc sinus surgery. 2. After infected foci have been removed or ruled out, it is most prudent to make every effort in restoring normal occlusion and to see that the bite is opened and balanced.

We should like to mention here that we have not yet had the opportunity of observing a case of Ménière's disease cleared up completely by the removal of other areas of infected foci, such as a tonsil or adenoid, gall bladder, appendix, prostate, lingual dorsum scraping, etc., as attested by Goodyear, Wright, Kemler, and others; nonetheless, we cannot deny the possibility of such occurrences.

The following case histories will illustrate our approach in the management of labyrinthine hydrops through the removal of infected oral foci and the maintenance of good temporomandibular function by opening the bite and balancing the occlusal planes.

Report of Cases

CASE 1.—A 35-year-old white woman had developed severe Ménière's attacks, at times more than twice weekly, four months previous to the initial examination by us. She reported her health as "excellent" up until the onset of her disease.

Examination revealed typical history, acoustical findings, and symptoms of Ménière's disease, together with rather severe dental disease, especially in the posterior teeth. Since the symptoms were right-sided, she was advised to have her upper molars and first and second bicuspids on this side removed. This was done, but with no alleviation of the attacks. She was then advised to have an alveolectomy in this area. This was done under local anesthesia, at which time the entire alveolar process was removed, as was the floor of the antrum. Some thickening of the mucous membrane of the antral floor was removed at the same time. The incision was sutured by interrupted 000 absorbable surgical sutures and irrigated at frequent intervals until healing was complete from the bottom out. The patient has been completely free of Ménière's attacks for two and a half years following this surgery.

CASE 2.—A white man, age 56, with severe Ménière's disease of about eight months' duration. This patient had consulted many physicians and clinics for help, but despite rather intensive treatment had not improved. Physical examination showed a normal white man except for typical symptoms of Ménière's disease. A positive tongue-blade test was elicited and a definite difference between the ramus of the mandible and maxilla on one side as compared to the other was noted. Though he was edentulous, dental x-rays revealed residual alveolar infections. An alveolectomy was performed on the affected right side. His severe attacks of dizziness disappeared immediately after surgery; however, tinnitus persisted, and on sudden change of position he complained of giddiness. No true vertigo was complained of after his surgery and he was able to drive his car. After several attempts at opening and balancing his bite, we were finally successful in completely alleviating the tinnitus and dizziness. Presently the tongue-blade test is no longer positive and the rami of the mandible appear equidistant from the body of the maxilla bilaterally.

CASE 3.—A 48-year-old white engineer whose history was insignificant up until the time of the onset of Ménière's disease two years previous to the time of our initial examination. He had been treated by eight or more physicians during this time and had consulted several large medical clinics in this country. In spite of intensive medical management and dietary restrictions, he was unable to perform his engineering duties. A complete upper posterior alveolectomy was done, followed by complete cessation of Ménière's attacks. Tinnitus persisted, but almost completely subsided after his bite was balanced by means of a partial dental plate. After four years he has had no recurrence of dizziness or vertigo and his hearing loss has stabilized.

CASE 4.—A 52-year-old white man, completely incapacitated from his work as a tax assessor 18 months previous to our examination. He had been treated medically by many physicians and had consulted two well-known clinics in the United States. When first seen by us he had the typical symptoms of Ménière's disease and a large laceration of the scalp, the result of a fall during a Ménière's attack in the railroad station on his way to the city. The patient also manifested a rather marked degree of chronic sinusitis of the left antrum and ethmoids. Four posterior upper teeth were removed and an alveolectomy was performed. The floor of the antrum was removed, and a large amount of pus and thickened membrane was removed from the antrum. A window was placed in the inferior meatus in the routine Caldwell-Luc fashion. This patient received immediate relief and has been free of his symptoms for four and one half years.

CASE 5.—A 49-year-old white male bus driver, who was involved in a traffic accident with his bus, was dismissed from his occupation seven months previous to our initial examination. He had been treated assiduously for Ménière's disease by several physicians and was advised to undergo an obliterative operation on the right labyrinth. Our examination showed the typical findings of Ménière's disease. Sinus x-rays revealed a marked clouding of the right antrum. A routine Caldwell-Luc procedure was done, with the removal of pus, polyps, and the entire mucous membrane of the antrum. A large window was placed in the inferior meatus, and drainage and healing occurred as expected. The patient experienced relief from his bothersome postnasal drip, but no relief whatever from his Ménière's attacks. Three months after the Caldwell-Luc procedure, an alveolectomy was performed with the removal of the entire antral floor on this side. It was observed that the window of the previous Caldwell-Luc procedure was well-healed and patent; however, the mucous membrane that had recently regenerated was again thickened at the antrum floor. This was removed again. Closure was done in the routine fashion with 000 plain absorbable surgical suture, and after frequent irrigations the incision healed with the patient experiencing no more Ménière's attacks. In a recent communication he reports no recurrence of symptoms in a period of more than seven years.

CASE 6.—A 33-year-old white housewife with a history of severe Ménière's disease for approximately two months. This patient was seen, diagnosed, and cared for by one of the leading clinics in the city. In spite of past hospitalization and intensive medical management, she had received no relief and was unable to perform her routine household duties. When she was seen by us it was

difficult to examine her because of her weakened condition and vertigo. There was considerable weight loss because of her inability to eat. She was again hospitalized and an alveolectomy performed. There was almost immediate relief from the severe vertigo and marked reduction in tinnitus. For a few days following surgery this patient would become slightly dizzy when walking down the corridor of the hospital if she watched the checkerboard pattern of floor tile colors. This symptom disappeared within a few days and the patient was discharged with marked improvement in over-all condition. Audiometric follow-up studies showed small but definite gains in hearing capacity, and a recent check shows that she has resumed all household duties and has been symptom-free for over one and a half years.

Case 7.—A 34-year-old white woman who had been treated for Ménière's disease for approximately 14 months. On different occasions she had been stopped by the police and charged with reckless driving which she reported was due to the onset of Ménière's attacks. Examination of this patient revealed typical findings of Ménière's disease. The tongue-blade test was positive and there was a marked relative spatial difference between the rami of the mandible and the maxilla on the two sides. Her teeth and sinuses appeared in adequate condition; however, deep fillings in the posterior teeth in both the upper and lower jaws were evident. She was referred to a dentist, who built a splint which opened and balanced her bite. She wore this appliance approximately three months but gained no relief from her Ménière's symptoms. The patient, very discouraged, consented to our suggestion of dental extraction. Her posterior upper teeth were removed, and a slight amount of the alveolar process was also removed. She gained immediate relief of her vertigo and moderate relief of her tinnitus. Audiometric studies indicate her hearing loss has persisted without change for the past three and one half years.

Case 8.—A 28-year-old white woman, wife of a dentist, who had her teeth extracted two years previously because of severe periodontoclasia. One year before our examination the patient suddenly began to have severe Ménière's attacks. She had been hospitalized three times and treated by several specialists. Our examination showed thickening of the antral mucous membrane which was not visible on a Water's view. She also elicited severe tenderness over the alveolar process on the left side posteriorly. Pressure over this area would cause the patient to become so dizzy she would fall. A severe nystagmus would develop at this time, and vomiting was not unusual. Though the dental arches seemed to be normal from an outward appearance, dental x-rays indicated areas suspect of residual infection. A rather extensive alveolectomy was done, and the thickened portion of the mucous membrane of the antrum was removed. The postoperative course was without incident, and the patient has been followed for over seven years with no recurrence of symptoms.

Case 9.—A 54-year-old white male office worker with a history of Ménière's disease for seven months previous to our examination. He lives in a large city and had had various types of medical treatment, including chiropractic adjustments. Attacks would occur two or three times a month and would incapacitate him for three or four days, after which he was able to return to work. He had stopped driving a car because of fear of accidents. Electroencephalograph studies had been made, and a complete set of sinus and dental x-rays had previously been taken. The latter he brought to our office. They had been interpreted as "normal"; however, on closer examination his dental pictures showed many deep fillings and two fixed bridges—one on either side of the upper posterior area. His dentist had assured him that all of his remaining teeth were vital and that his trouble could not be dental in origin. His posterior teeth were removed, along with the bridgework, and a rather complete alveolectomy was done. The patient received immediate and complete relief from his trouble and has worked over five years without a recurrence of symptoms and loss of office time.

Case 10.—A 48-year-old white male sheep herder from the Northwestern United States. This man had been bothered by attacks of dizziness, but not true vertigo for about a year. He was advised not to take his herd of sheep to the mountains for grazing because of his illness. Despite numerous examinations by various physicians, no reason for these attacks was found. The patient was and had been a tobacco chewer for many years and his teeth were worn down markedly, even though he had not a single cavity. On examination the tongue-blade test was markedly positive and would elicit attacks of dizziness instantaneously. He also exhibited severe tenderness over the tuberosity on both sides and over the anterior border of the ramus of the mandible bilaterally. An onlay splint was designed, and the bite was opened about 5 mm. The patient was advised to wear this appliance at all times, and he did. He received almost immediate relief from his attacks of dizziness. Six months after he started to wear the appliance he broke it accidentally and was without it for a week while repairs were being made to it. After five days without the appliance his attacks recurred. They were stopped when he started to wear the appliance again. In the interim of seven years he has had no trouble with his original complaint, but has had three additional appliances made because

of their frequent wearing down and his fear that bite changes would start his old trouble anew.

Colloidal Basis of Hydrops Physiology

It becomes necessary at this point of our discussion to attempt an explanation of the mechanism and physiology of endolymphatic hydrops. Since Young's findings give us an anatomical structure upon which to build, we can superimpose upon it that which is already known in medical science.

It is known that chemical substances put in the human body very often show an intricate degree of selectivity and affinity for specific tissues. Thus streptomycin shows a very specific affinity for vestibular structures; and dihydrostreptomycin shows specific predilection to the cochlear structures. We mention this to illustrate the fact that bacterial toxins too can also be very specific in their toxicity. Diphtheria toxin, for example, is known to attack the myocardium; and Gonococcus attacks the valves of the right side of the heart. From the viewpoint of focal infection as the etiological basis of Ménière's disease, then, it is not a chimerical position to hold that microscopic organisms or toxins from a focus of infection can produce toxic changes in the labyrinth which may, and do, account for some of the problems we encounter. We believe, therefore, that Lempert, Wolff, et al.'s observations of herpetic lesions and contamination in the labyrinth are accurate, and that they are of toxic rather than trophic origin.

To refute or corroborate evidence set forth by Williams, Hallpike, Mygind and Dederding, Perlman et al., Lindsay, Hilger, Kemler, Derlacki, Hanson, Fowler, and others, we maintain that localized edema or hydrops can, and usually does, occur as the result of tissue invasion by bacteria or bacterial toxins showing specific selectivity for labyrinthine structures. This phenomenon is observed almost invariably in every disease known in medicine, and further explanation would be an elaboration of the obvious. Furthermore, to assume that the symptoms encountered in Ménière's disease are the result of a localized imbalance of the sympathetic or parasympathetic nervous system appears cumbersome in the light of basic physiology. If one must assume, it would be more realistic to assume the nervous system reacting in an orderly manner, but that the structures themselves are edematous because they are striving to maintain a balanced colloidal environment. Mygind and Dederding's assumptions of the significance of water metabolism from their ear experiments, we feel are precarious, since they are based on Starling's earlier work on water retention in animal and plant tissue. Starling's conclusions, as later experimentation has demonstrated, are fraught with unresolved inconsistencies. We therefore subscribe to the colloidal concept of fluid absorption as advanced by Ostwald and others, and maintain that what is observed in an endolymphatic hydrops is the uptake of fluid due to the presiding hydrophilic colloid which has increased its water-holding capacity owing to bacterial toxins or bacteria. (See Fischer on "Edema & Nephritis"; and Fischer and Hooker on "The Lyophilic Colloids.")

Allergy Explanation Untenable

As to the hypothesis of allergy causing Ménière's disease, the statistics cited earlier by Cawthorne and Hewlett indicate this to be untenable in over 90% of 900 cases analyzed by them. In this regard, we might mention that it is difficult, if not impossible, to explain one unknown by another unknown. And since allergy is a state of pathology that no one understands from the biochemical standpoint, it must occupy a rather tenuous etiological status in regard to Ménière's disease. As one medical writer aphoristically, if not accurately, stated: "When we are faced with an unexplainable situation, we invent a term, and surely 'allergy' is such a term."

Perhaps the critics of the focal infection-temperomandibular bite-change theory in Ménière's disease will, in rebuttal, refer to "focal infection" as such a term also.

Nevertheless, we feel that this approach, interpreted in the concepts as outlined above, reconciles most, if not all, of the conflicting evidence heretofore presented by previous investigators. Moreover, to us, if to no one else, it logically accounts for the various physiological and audiological symptoms evidenced in this distressing ailment.

Summary and Conclusions

In summary, we are submitting a confirmation of the etiological concept of focal infection in Ménière's disease as advanced by previous writers. We maintain (1) that endolymphatic hydrops is the result of changes in the water-holding capacity of the colloidal structures due to toxins or organismic invasion, specifically infected foci; (2) that the infected foci predominate within the structures of and around the teeth and/or sinuses, but that other areas of infection (tonsils, tongue, adenoids, gall-bladder, etc.) may be causative in some cases; (3) that Young's work supports the focal infection theory by presenting an anatomical framework showing lymphatic pathways upon which organisms and/or toxins could travel; (4) that routine sinus x-rays and diagnostic irrigations often fail to show infection when such is present; (5) that infected foci are not necessarily removed by routine teeth extractions and/or Caldwell-Luc procedures; (6) that bite problems often introduce a variable associated with the tempero-mandibular joint complicating the true symptoms of Ménière's disease and necessitating establishment of normal dental occlusal planes; (7) that removal of infected foci attacks the cause of labyrinthine hydrops and as such must be considered superior to the symptomatic therapeutic approaches.

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BIBLIOGRAPHY

Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. 116:1753-1760 (April 19) 1941.

Blum, N. W.: Radiographic Interpretation, Am. Dent. Surgeon 46:123-124, 1926.

Facts Versus Opinions, Am. Dent. Surgeon 46:248-251 (March) 1926.

Diagnosis and Treatment of the Maxillary Sinus: Its Relation to the General Dental Practitioner, Am. Dent. Surgeon 51:285-291 (Nov.) 1931.

Björk, H.: Dental Sinusitis: Frequency and Treatment, Transactions of 12th Nordic Otolaryngological Congress, Acta Oto-laryng., Supp. 118, pp. 45-53, 1954.

Cawthorne, T., and Hewlett, A. B.: Ménière's Disease, Proc. Roy. Soc. Med. 47:663-670 (Aug.) 1954.

Costen, J. B.: A Syndrome of Ear and Sinus Symptoms Dependent upon Disturbed Function of the Temperomandibular Joint, Ann. Otol. Rhin. & Laryng. 43:1-15 (March) 1934.

Neuralgias and Ear Symptoms Associated with Disturbed Function of the Temperomandibular Joint, J. A. M. A. 107:252-255 (July 25) 1936.

Diagnosis of Mandibular Joint Neuralgia and Its Place in General Head Pain, Ann. Otol. Rhin. & Laryng. 53:655-659 (Dec.) 1944.

Crowe, S. J.: Ménière's Disease: A Study Based on Examinations Made Before and After Intracranial Division of the Vestibular Nerve, Medicine 17:1-36 (Feb.) 1938.

Day, K. M.: Labyrinth Surgery for Ménière's Disease, Larygoscope 53:617-630 (Oct.) 1945. (April) 1945.

Ménière's Disease: Present Concepts of Diagnosis and Management, Ann. Otol. Rhin. & Laryng. 59:966-979 (Dec.) 1950.

Derlacki, E. L.: Non-Surgical Management of Ménière's Disease, Laryngoscope 64:271-284 April) 1954.

Feinberg, S. M.: Allergy in Practice, Ed. 2, Chicago, The Year Book Publishers, Inc., 1946.

Fischer, M. H.: Oedema and Nephritis, Ed. 3, New York, John Wiley & Sons, Inc., 1921.

Fischer, M. H., and Hooker, M. O.: The Lyophilic Colloids (Their Theory and Practice), Springfield, Ill., Charles C Thomas, Publisher, 1933.

Fowler, E. P.: Tinnitus, Deafness, and Vertigo, A. M. A. Arch. Otolaryng. 64:196-204 (Sept.) 1956.

Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ménière's Symptom Complex: Medical Treatment, Ann. Otol. Rhin. & Laryng. 43: 1035-1046 (Dec.) 1934.

Goodyear, H. M.: Ménière's Disease: Simplified Surgical Management, Ann. Otol. Rhin. & Laryng. 63:491-497 (June) 1954.

Ménière's Disease, A. M. A. Arch. Otolaryng. 63:343-346 (April) 1956.

Green, R. E., and Douglass, C. C.: Intracranial Division of the Eighth Nerve for Ménière's Disease: Follow-Up Study of Patients Operated on by Dr. Walter E. Dandy, Ann. Otol. Rhin. & Laryng. 60:610-621 (Sept.) 1951.

Gundrum, L. K.: Etiologic Analysis of 100 Cases of Ménière's Symptom-Complex, A. M. A. Arch. Otolaryng. 57:123-128 (Feb.) 1953.

Hallpike, C. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, Proc. Roy. Soc. Med. 31:1317-1336 (Sept.) 1938.

———and Harrison, M. S.: Ménière's Disease Treated by Portmann's Operation, A. M. A. Arch. Otolaryng. 60:141-144 (Aug.) 1954.

Hanson, H. V.: Treatment of Endolymphatic Hydrops (Ménière's Disease) with Streptomycin, Ann. Otol. Rhin. & Laryng. 60:676-691 (Sept.) 1951.

Hargrove, S. W. G.: Treatment of Oro-Antral Fistulae, J. Laryng. & Otol. 69:709-728 (Nov.) 1955.

Hilger, J. A.: Autonomic Dysfunction in the Inner Ear, Laryngoscope 59:1-11 (Jan.) 1949.

———and Goltz, N. F.: Some Aspects of Inner Ear Therapy, Laryngoscope 61:695-717 (Aug.) 1951.

Hoople, G. D.: Symposium, Ménière's Disease: Medical Treatment, Tr. Am. Acad. Ophth. 60:167-198 (March-April) 1956.

Johnson, L. F.: Surgery of the Sympathetic in Ménière's Disease, Tinnitus Aurium, and Nerve Deafness: New Concept in Acute Fulminating Ménière's Disease, A. M. A. Arch. Otolaryng. 59:492-498 (April) 1954.

Kemler, J. L.: Influence of Focal Infection on Ménière's Disease, Eye, Ear, Nose, & Throat Month. 32:697-700 (Dec.) 1953.

Recent Concepts Concerning Ménière's Syndrome, Eye, Ear, Nose, & Throat Month. 35: 781-784 (Dec.) 1956.

Lempert, J.; Wolff, D.; Rambo, J. H. T.; Wever, G. E., and Lawrence, M.: New Theory for the Correlation of the Pathology and the Symptomatology of Ménière's Disease, Ann. Otol. Rhin. & Laryng. 61:717-746 (Sept.) 1952.

Lillie, H. I.; Horton, B. T., and Thornell, W. C.: Ménière's Symptom Complex: Observations on Hearing of Patients Treated with Histamine, Ann. Otol. Rhin. & Laryng. 53:717-741 (Dec.) 1944.

Lindsay, J. R.: Labyrinthine Dropsy and Ménière's Disease, Arch. Otolaryng. 35:853-867 (June) 1942.

Labyrinthine Dropsy, Laryngoscope 56:325-341 (July) 1946.

Ménière's Disease: Differential Diagnosis and Treatment, Manual Prepared for Graduates in Medicine, Rochester, Minn., American Academy of Ophthalmology and Otolaryngology, 1947.

Present Status of Ménière's Disease: Pathology, in Symposium, American Academy of Ophthalmology and Otolaryngology; abstracted, Mod. Medicine, pp. 135-139, Sept. 15, 1956.

McKenzie, K. G.: Intracranial Division of the Vestibular Portion of the Auditory Nerve for Ménière's Disease, Canad. M. A. J. 34:369-381 (April) 1936.

Meyer, O.: Residualinfektionen nach Zahnertraktionen und Mandeloperationen, Med. Klin. 44:952-955 (July 29) 1949.

Murray, M.: Alveolectomy: Its Indications and Technique, Ohio M. J. 44:1220-1221 (Dec.) 1948.

———and O'Ferrell, E.: A Bacteriological Check on Residual Dental Infection, J. Lab. & Clin. Med. 27:154 (June) 1942.

Mygind, S. H., and Dederding, D.: Significance of Water Metabolism in General Pathology as Demonstrated by Experiments on the Ear, Acta Oto-laryng. 17:424-426, 1932.

The Diagnosis and Treatment of Ménière's Disease, Ann. Otol. Rhin. & Laryng. 47:768-774 (Sept.) 1938.

The Pathogenesis of Ménière's Disease and of Kindred Conditions in the Ear and the Rest of the Body, Ann. Otol. Rhin. & Laryng. 47:938-946 (Dec.) 1938.

Ostwald, C. W. W.: Handbook of Colloid Chemistry, Philadelphia, P. Blakiston's Son & Co., 1919.

An Introduction to Theoretical and Applied Colloid Chemistry, Ed. 2, New York, John Wiley & Sons, Inc., 1922.

Passe, E. R.: Sympathectomy in Relation to Ménière's Disease, Nerve Deafness and Tinnitus: Report on 110 Cases, Acta Oto-laryng. 42:133-151 (Feb.-March) 1952.

Perlman, H. P.; Goldinger, J. M., and Coles, J. O.: Electrolyte Studies in Ménière's Disease, Laryngoscope 63:640-651, 1953.

Portmann, G.: The Saccus Endolymphaticus and an Operation for Draining the Same for the Relief of Vertigo, J. Laryng. & Otol. 42:809-817 (Dec.) 1927.

Reading, P.; Harrison, D. F. N., and Dinsdale, R. C. W.: The Causation, Pathology, and Treatment of Ora-Antral Fistulae Resulting from Dental Extraction, J. Laryng. & Otol. 69:729-743, 1955.

Rosen, S.: Surgery in Ménière's Disease, Ann. Otol. Rhin. & Laryng. 60:657-667 (Sept.) 1951.

Selfridge, G.: A Survey of the Relation Between Nutrition and the Ear, Ann. Otol. Rhin. & Laryng. 49:674-687 (Sept.) 1940.

Shambaugh, G. E., Jr.: Histamine in Treatment of Certain Types of Headaches and Vertigo Following the Fenestration Operation, Arch. Otolaryng. 51:781-785 (June) 1950.

Sheldon, C. H., and Horton, B. T.: Treatment of Ménière's Disease with Histamine Administered Intravenously, Proc. Staff Meet. Mayo Clin. 15:17-21 (Jan.) 1940.

Strong, M. S.: Dorsal Sympathectomy in Labyrinthine Disease, A. M. A. Arch. Otolaryng. 65:340-348 (April) 1957.

Sylvester, H. M.: Die Technik der radikalen Alveolektomie, Deutsche. Zahnartl. Ztsch. 6: 314-319 (March 15) 1956.

Thompson, J. R.: Temporomandibular Disorders: Diagnosis and Treatment, in The Temporomandibular Joint, edited by B. G. Sarnat, Springfield, Ill., Charles C Thomas, Publisher, 1951.

Vaughan, W. T.: Practice of Allergy, The C. V. Mosby Company, St. Louis, 1939.

Walsh, T. E.: Diagnosis and Treatment of Ménière's Disease, A. M. A. Arch. Otolaryng. 64:118-128 (Aug.) 1956.

Weinmann, J. P., and Sicher, H.: Pathology of the Temporomandibular Joint, in The Temporomandibular Joint, edited by B. G. Sarnat, Springfield, Ill., Charles C Thomas, Publisher, 1951, Chap. 4.

Williams, H. L.: Ménière's Disease, Springfield, Ill., Charles C Thomas, Publisher, 1952.

Wright, A. J.: Ménière's Disease, Proc. Roy. Soc. Med. 41:801-805, 1948.

Young, M. W.: The Drainage of the Perilymph in Macacus Rhesus, read at the 62d Annual Session of the American Association of Anatomists; abstracted, Anat. Rec. 103:524, 1949.

Anatomical Basis for Labyrinthine Disturbances Resulting from Dental Deformities, read at the 63d Annual Session of the American Association of Anatomists; abstracted, Anat. Rec. 106:296, 1950.

Metastasis from Glomus Jugulare Tumors

Discussion of Nomenclature and Therapy

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Frequently, I [1] have pointed out how limited is the experience of even the most experienced among us with regard to the knowledge and behavior of glomus jugulare tumors and how unwise it is to be dogmatic or arbitrary with respect to some of the unsolved clinical problems: I. Metastasis: How frequently does it occur and to what areas? II. Nomenclature: How shall these tumors ultimately be designated? III. Therapy: What can be adjudged a rational method of treatment?

I. Metastasis

The time has come, I believe, to examine the concept that carotid-body-like tumors, or by whatever term one designates these tumors, are benign and do not metastasize. In 1954, Figi and Weisman [2] were of the opinion that those instances of metastasis reported in the literature were probably coincidental occurrences of chemodectoma and carotid-body tumors. Lattes and Waltner [3] in 1949 reported what they regarded as a metastasis of a nonchromaffin paraganglioma to the liver, admitting, however, that their sections, altered perhaps by postmortem changes, showed poor preservation of tissue. Williams [4] in 1955, commenting on metastasis, said since metastasis has not been reported in any of the later cases in this country and abroad, he was inclined to regard the report of Lattes and Waltner with reasonable doubt. He felt the consensus of most observers was that these tumors were malignant only because of their possible encroachment on the intracranial structures, that they did not metastasize, and, therefore should be classified as benign neoplasms. Gaffney [5] in 1953, in discussing the spread of these tumors (*a*) to regional lymph nodes (Winship, Klopp, and Jenkins,[6] 1948); (*b*) to liver (Lattes and Waltner,[2] 1949); (*c*) to liver and lungs (Tamari, McMahon, and Bergendahl,[7] 1951); and (*d*) to liver, lungs, and spleen (Henson, Crawford, and Cavanagh,[8] 1953), questioned the accuracy of the observations of Lattes and Waltner and of Tamari et al. According to Gaffney, these tumors were nonmetastasizing though locally very invasive.

Glomus jugulare tumors often will follow a consistent clinical pattern in their growth and extent. From the majority of clinically reported instances one would be correct in regarding these tumors as benign, in that they often are slow growing, causing local destruction, and do not contain mitotic figures. Riemenschneider et al.,[9] Capps,[10] and others have suggested that these tumors be classified into two categories: (*a*) those which remain confined to the middle ear for a long time, causing deafness and tinnitus, but do not destroy the tympanic membrane; (*b*) those others which probably arise from glomus structures in the jugular bulb region and involve the middle ear, the petrous pyramid, and the intracranial cavity. This latter group some term malignant because it is so invasive locally. It is worthy of note that although, clinically, these must be regarded as malignant, sections of the tumors again do not show mitotic figures and other cellular changes commonly noted in malignant tumors. There is increasing data to controvert the attitude that these tumors must be regarded as benign.

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Recently, Stewart, Ogilvie, and Sammon [11] stated that metastasis did not occur in their series. They further declared that glomus jugulare tumors rarely killed and that many cases go through life with comparatively little discomfort. On the other hand, they also state that tumors of the glomus jugulare and paraganglion juxtavagale are clinically malignant, the characteristic feature being local, albeit often extensive, invasiveness. They suggest that the best form of treatment is local removal of the growth combined with radiotherapy. Analysis of their cases disclose that the tumor dose of radiation employed was 3280-3920 r with optimum skin dose of 4000-5000 r delivered in a four-week period. Poulsen [12] (1956), in discussing glomus jugulare tumors, also stated that metastases are rare and emphasized local invasiveness.

II. Nomenclature

Otolaryngologists, neurosurgeons, and pathologists have displayed tremendous interest in this tumor in recent years. A manifestation of this interest has resulted in considerable confusion insofar as a uniform nomenclature is concerned. When Otani and I [13] were first confronted with this tumor we were at a loss as to a fitting name and tentatively suggested the term carotid-body-like tumor, because of the strikingly identical morphology with known carotid-body tumors in the neck. We were well aware that this term was not an ideal one in the sense that the term did not fulfill the accepted tradition in pathology in describing a tumor.

In rapid succession the tumor was termed glomus jugulare tumor (Winship),[6] which is, in my opinion, a good term because it signifies that this is a tumor of the glomus jugulare. It has a certain advantage in that it indicates a tumor of the glomus structure which is located in the jugular bulb region. Shortly thereafter, Lattes and Waltner [2] reported a series of similar cases and offered what they considered a more descriptive name, nonchromaffin paraganglioma. It is true that these tumors, thus far, have proved to be nonchromaffin-staining tumors. It is also admitted that they resemble paragangliomata, but as yet there is no evidence to indicate that these structures possess paraganglionic function or any other function. Mulligan [14] suggested the name chemodectoma for these tumors because he felt that this was an aptly descriptive term for any neoplasm arising from paraganglionic tissue. He proffered this term, assuming that the cells were receptor cells, which are often associated with the distribution of parasympathetic nerves which originate in the adventitia of blood vessels and in structures intimately connected with afferent nerve fibers or along the branches or in the ganglia of the glossopharyngeal nerve. Gaffney [5] did not feel that glomus jugulare, the carotid and aortic bodies should be classified with the histologically similar paraganglia. He agreed that they might be derived from neural crest primordia. On the other hand, he felt there were other desiderata which preclude them from being considered paraganglionic. First, their innervation appears to be mainly sensory. Secondly, neither they nor the tumors arising from them have been shown conclusively to secrete a pressor substance. Thirdly, they are nonchromaffin-staining. On the basis of this Gaffney felt that these structures were not glomus, and almost certainly not paraganglionic, and that, if it be accepted that they are developmentally related to the carotid body, then their function, if they have one, is probably that of a chemoreceptor organ, and thus these neoplasms should be regarded as receptomas. Zettergren and Lindström [15] referred to these tumors as glomerocytomas. Lundgren [16] applied the term tympanic body tumors, a term which, while descriptive of the location, falls far short of an acceptable morphological nomenclature.

It is, therefore, apparent that to bring order out of this confusion, efforts must be made to determine the function of the glomus structures in the middle ear, inner

tympanic wall, and jugular bulb area. When this knowledge is established, the suitable pathological designation for this tumor will be evident. It is understandable that one should deduce similar physiological function for tissue that has an identical histologic and morphologic structure. This is a deduction and not scientific proof.

III. Therapy

I am of the opinion that a rational mode of therapy can logically be recommended if one attempts to classify the stages in which one sees these tumors. Many observers have been impressed with the relative ease in coping with these tumors when they are small and confined to the middle ear. Aids in early diagnosis have been proposed by Brown,[17] who attempted to elicit a pulsation sign in instances of localized tumor behind an intact drum, and by Bradley and Maxwell,[18] who called attention to the potential significance of a discolored tympanic membrane associated with a conductive deafness in early diagnosis. In my opinion any aids in establishing a diagnosis early when the tumor is small and confined to the middle are consonant with the best possible prognosis. Rational therapy in these instances would be elevation of the drum and complete ablation before the hearing is seriously impaired, and before local invasion of the adjacent important structures takes place. Secondly, in the instances in which the tumor has eroded the tympanic membrane and the mastoid bone, and secondary infection has taken place, often presenting the picture of middle ear suppuration, then radical mastoidectomy is indicated. If it is possible to remove all of the tumor, I withhold irradiation. If it is doubtful that all the tumor is resectable, I follow with irradiation, preferably cobalt therapy, if it is available. Thirdly, when the patient is seen late in the course of the disease with obvious extensive involvement of the mastoid and the petrous bone and with intracranial involvement, the patient's interests will be served best by resorting to irradiation rather than to extensive multilating surgery.

One must realize that for many reasons it is most difficult to evaluate a modality of therapy in glomus jugulare tumors, whether it be surgery or irradiation, particularly when it is generally known that these tumors, untreated, have at times been known to change little in 15-20 years. However, recent clinical experiences have made me aware that at times these tumors may behave in a manner which would entirely controvert such a complacent attitude with regard to ultimate prognosis and treatment. In order that our knowledge of these tumors be augmented, two recent clinical experiences are herewith recounted.

Report of Cases

CASE 1.—Patient, a man aged 55 years, was admitted to The Mount Sinai Hospital, Feb. 24, 1955, with a two months' history of pain in the right ear. There was no discharge. The following day, a right peripheral facial paralysis developed. It was also noted that the right palpebral fissure was wider than the left and that there was a tendency for the right eye to cross.

Pain continued intermittently in the right ear, and the facial paralysis was unchanged. One month before admission the patient was treated for an inflamed ear with chlortetracycline (Aureomycin) and ear drops. The next day there was dizziness, and double vision was noted for the first time. The patient then began to vomit. Vomiting and dizziness persisted for three days, and then subsided. The pain persisted; facial paralysis became progressively worse, and one week prior to admission the onset of localized low-back pain, slowly increasing in severity, was noted. For five days he noted progressive difficulty in swallowing.

Examination revealed an acutely ill man with blood pressure 140/80, pulse 80, respirations 16, temperature 100 F. There was a right peripheral facial weakness, and right nerve deafness. The pupil on the right side was dilated and unreactive. The uvula and palate were deviated to the left. There was a slight weakness in the right lateral conjugate gaze, a mild unsteadiness in gait, and deep tendon reflex preponderance on the left. The right external canal revealed a vascular-appearing tumor with an excoriated surface. There was a scant, pulsating discharge, and the short process of the malleus was clearly visible.

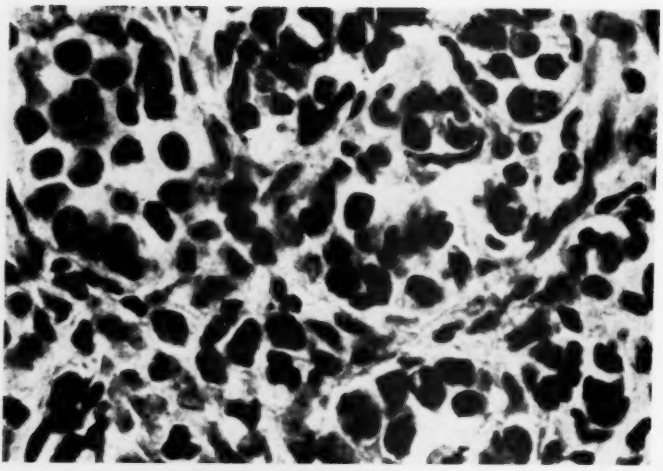

Fig. 1. — Microphotograph showing the typical cell nests surrounded by capillaries. The cells show more irregularity than the usual classic pattern.

Fig. 2.—Roentgenogram of the chest showing an elliptical mass in the right axillary region with destruction of the sixth rib.

Laboratory data were essentially negative. Biopsy of the mass in the canal was attendant with profuse bleeding. Pathological study of biopsy specimen was reported as "paraganglioma" (Fig. 1).

X-ray of mastoids revealed "the right mastoid to be of the sclerotic variety with only a small amount of cellular structure posteriorly. There is no evidence of bone destruction."

X-ray of skull: "Sella turcica, dorsum sellae, and clinoid processes appear normal. Pineal body is calcified and not displaced. The sphenoid wings are not unusual. The right frontal sinus is small but there is no abnormality in sinuses."

X-rays of chest (Figs. 2 and 3) reveal "an elliptical mass in the right axillary region with destruction of the 6th rib in this area. The remainder of the lung field appears normal."

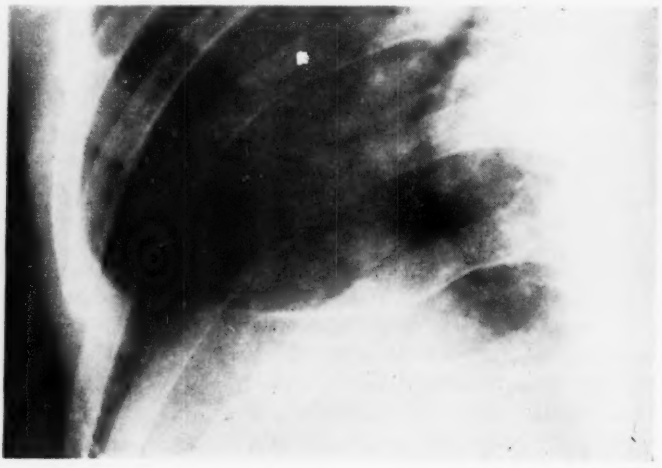

Fig. 3.—Lateral roentgenogram of the chest showing destruction of the sixth rib on the right side.

Fig. 4.—Roentgenogram of the spine showing erosion of the anterior aspect of the 10th dorsal vertebra with increased sclerosis of the inferior half.

X-ray of the dorsal spine (Fig. 4): "The destroyed rib on the right side is again noted on the lateral view. There is also some increased sclerosis of the inferior half of the 10th dorsal vertebra as well as some erosion of the anterior aspect of this vertebra.

Clinical course in the hospital revealed a tumor involving the right middle ear with extension to the intracranial cavity. There was clinical and x-ray evidence of metastases to the sixth rib and dorsal spine. The patient was discharged to his home for follow-up care and therapy. Despite irradiation he went rapidly down hill and died within six months.

CASE 2.—A woman patient aged 53 years complained of tinnitus in the right ear, for which little explanation could be found. On her second hospital admission in 1951, a mass was noted in the floor of the nose and involving the palate on the right

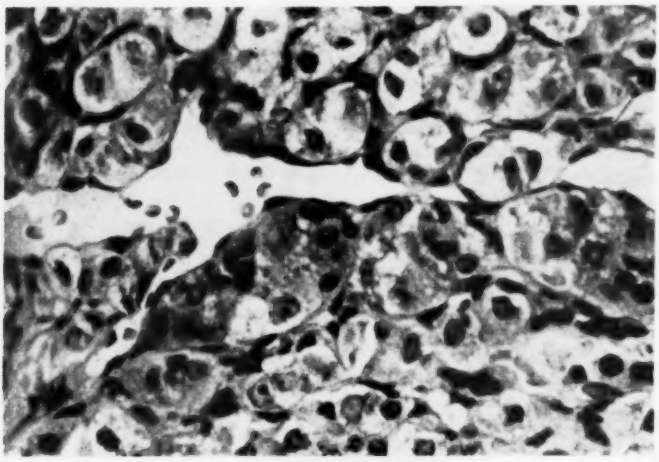

Fig. 5. — Photomicrograph showing the classical pattern of carotid-body-like tumor, showing epithelioid cell nest formation separated by capillaries.

Fig. 6. — Anteroposterior view of the spine showing marked collapse of the body of the seventh dorsal vertebra. Disk interspaces above and below are well preserved.

Fig. 7.—Roentgenogram showing lateral view of the spine revealing marked collapse of the body of the seventh vertebra.

side. A biopsy was done and was reported as a typical carotid-body-like tumor. She was treated with standard 200 kv. type radiation, the total dosage being 6500 r. In 1954, the patient was readmitted to the hospital for the third time because of increasing tinnitus and what appeared to be a bluish coloration, suggesting hemorrhage in the right middle ear. Biopsy specimen from the posterior portion of the nose in the nasopharynx, right, was taken and again proved positive for carotid-body-like tumor (Fig. 5). The patient was again treated with irradiation, receiving approximately 6500 r.

The right tympanic membrane was elevated; the middle ear was carefully inspected and found free of tumor. The patient was seen by our consulting orthopedist because of severe back pain. There was local tenderness, and the spine was x-rayed. In view of our previous experience, the chest was x-rayed despite the absence of symptoms referable to the chest. There was a suspicious change in one rib.

The AP view of the spine showed marked collapse of the seventh dorsal vertebra (Figs. 6 and 7). The disk interspaces above and below are well preserved. The lateral view clearly shows the marked collapse of the body of the seventh vertebra.

In view of x-ray findings and the localized pain and tenderness over this area, an aspiration biopsy was made from the suspicious seventh dorsal vertebra. It revealed typical histologic characteristics of metastatic glomus jugulare tumor (Fig. 8).

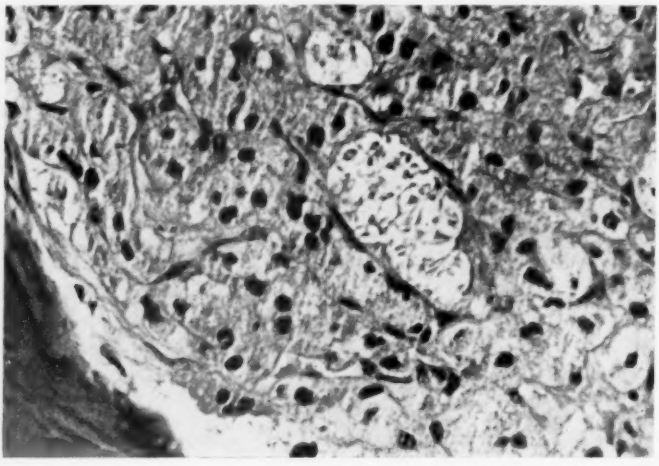

Fig. 8.—Aspiration biopsy specimen from the seventh dorsal vertebra showing typical histologic characteristics of carotid-body-like tumor.

The futility of surgery was readily evident, and the patient was treated with irradiation. The total amount this patient received to the nasopharynx was 13,650 r in air and the total skin dose was 16,000 r. The calculated tumor dose is 7000 r in 52 days, and the calculated dose administered at D-7 was 2300 r in 21 days. This excessive dosage was required because of the rapid extension of the tumor after the initial 6500 r to the floor of the nose. The extreme variability in the sensitivity to x-rays is graphically demonstrated by the good results reported by Williams et al. with dosages averaging 2000 to 3000 r, in contrast to the above-described clinical experience, in which it was not possible to control the growth of tumor with 13,000 r delivered in two courses of therapy, a year apart.

Summary

The incidence of metastasis from glomus jugulare tumors will be determined in the future, as the awareness of the possibility of metastasis stimulates the search for metastatic foci. Two cases are herewith reported to record that these tumors have metastasized to the dorsal spine and to the ribs.

The nomenclature of this interesting tumor has been briefly discussed in the hope that this discussion will dispel some of the confusion resulting from the use of so many different names in reporting the same tumor.

Therapy predicated upon surgical accessibility commends itself to me. Complete surgical removal is possible when the tumor is small. When the tumor involves the middle ear and mastoid area, complete extirpation is not always attainable. Radical mastoidectomy combined with irradiation is then the method of choice. When the tumor has invaded the petrous pyramid with evidence of jugular foramen syndrome, the treatment of choice is irradiation.

65 E. 76th St. (21).

REFERENCES

1. Rosenwasser, H.: Glomus Jugulare Tumor of Middle Ear, Laryngoscope 62:623-633 (June) 1952.

2. Figi, F. A., and Weisman, P. A.: Cancer and Chemodectoma of the Middle Ear and Mastoid, J. A. M. A. 156:1157-1162 (Nov. 20) 1954.

3. Lattes, R., and Waltner, J. G.: Nonchromaffin Paraganglioma of Middle Ear, Cancer 2:447-468 (May) 1949.

4. Williams, H. L.; Childs, D. S., Jr.; Parkhill, E., and Pugh, D. G.: Chemodectoma of the Glomus Jugulare, with Special Reference to Their Response to Roentgen Therapy, Tr. Am. Otol. Soc., 1955, pp. 264-290.

5. Gaffney, J. C.: Carotid-Body-like Tumors of Jugular Bulb and Middle Ear, J. Path. & Bact. 66:157-170 (July) 1953.

6. Winship, T.; Klopp, C. T., and Jenkins, W. H.: Glomus-Jugularis Tumors, Cancer 1:441-448 (Sept.) 1948.

7. Tamari, M. J.; McMahon, R. J., and Bergendahl, E. H.: Carotid Body-like Tumors of Temporal Bone with Particular Reference to Glomus Jugulare Tumors, Ann. Otol. Rhin. & Laryng. 60:350-364 (June) 1951.

8. Henson, R. A.; Crawford, J. V. and Cavanagh, J. B.: Tumors of Glomus Jugulare, J. Neurol. Neurosurg. & Psychiat. 16:127-138 (Aug.) 1953.

9. Riemenschneider, P. A.; Hoople, G. D.; Brewer, D.; Jones, D., and Ecker, A.: Roentgenographic Diagnosis of Tumors of Glomus Jugularis, Am. J. Roentgenol. 69:59-65 (Jan.) 1953.

10. Capps, F. C. W.: Treatment of Glomus Jugulare and Petrous or Tympanic Body Tumors of Middle Ear Bone with a Particular Reference to Radiotherapy, abstracted Excerpt Med. XI, 6:1046, 1953.

11. Stewart, J. P.; Ogilvie, R. F. and Sammon, J. D.: Tumours of the Glomus Jugulare and Paraganglion Juxtavagale of the Ganglion Nodosum, J. Laryng. & Otol. 70:196-239 (April) 1956.

12. Poulsen, I. P. V.: Middle Ear Tumours Proceeding from the Glomus Jugulare, Acta Otolaryng. 46:58-63 (Jan.-Feb.) 1956.

13. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid, Arch. Otolaryng. 41:64-67 (Jan.) 1945.

14. Mulligan, R. M.: Chemodectoma in the Dog, Am. J. Path. 26:680-681 (July) 1950.

15. Zettergren, L., and Lindström, J.: Glomus Tympanicum, Acta path. et. microbiol. scandinav. 28:157-164, 1951.

16. Lundgren, N.: Tympanic Body Tumours of Middle Ear, Acta oto-laryng. 37:367-379, 1949.

17. Brown, L. A.: Glomus Jugulare Tumor of Middle Ear, Laryngoscope 63:281-292 (April) 1953.

18. Bradley, W. H., and Maxwell, J. H.: Neoplasms of Middle Ear and Mastoid, Laryngoscope 64:53-556 (July) 1954.

Treatment of Complicated Colds with Acellular Bacterial Antigen Complex

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The common cold [1,2] is our greatest single cause of illness. Although the common cold has a specific viral etiology, its most frequent complications are bacterial. These bacterial complications—sinusitis, pharyngitis, laryngitis, tracheitis, bronchitis, bronchiectasis, and infectious asthma—follow the growth of the common Gram-positive pathogens,[2] particularly Staphylococcus aureus.

Recurrent upper respiratory infections are the largest single cause [3] of all diseases treated in general practice and pediatrics. These infections do not remain localized in the ear, nose, throat, and bronchi, but become systemic with serious complications.

Serious bronchopulmonary complications in children,[4] such as bronchiectasis and infectious asthma, can frequently be accounted for by some previous acute respiratory infection or a series of such infections, frequently repeated. These children [4] in their medical history show evidence of an inherited tendency to allergy, an acquired sensitivity to bacteria, and an unstable autonomic nervous system.

In adults who are highly susceptible to secondary infections or give a history of repeated and prolonged colds one often sees serious bacterial complications. In these patients the common cold is but the forerunner of serious bacterial complications such as sinusitis, pharyngitis, laryngitis, tracheitis, bronchitis, bronchiectasis, and infectious asthma.

Prigal [5] estimates that 1,250,000 Americans have sinusitis; 2,000,000 suffer from bronchitis, and another 1,000,000 suffer from infectious asthma. He emphasizes that the "problem is not the control of specific incidents of infection but how to prevent the recurrence of infection since this seemed to be inevitable in most patients." Further Prigal [5] states that "these repeated infections frequently starting in early infancy burden the patient with disability for many years if not an entire lifetime."

In recent years the treatment of these repeated complicated colds has been chiefly with antibiotics. This era of intense use of antibiotics has unfortunately detracted from the necessary study of antigens useful in the treatment of patients with recurrent respiratory infections and their devastating sequelae. This omission is an even more serious one when the evidence of bacterial allergy in these patients is taken into account.

Antibiotic therapy has not solved the problem of recurrent upper respiratory infections. The indiscriminate use of antibiotics [6] has interfered with immune-body production and has been of no value in producing desensitization to bacteria or their products. Allergic reactions [7] to antibiotics have become a serious problem, and even deaths are reported following their use. Such therapy has resulted in the development of resistant strains of bacteria [8] and in some patients in the overgrowth of a normally depressed bacterial flora, which then become a greater menace to the patient's health. The problem of antibiotic resistant pyogenic micrococci in hospitalized patients [9] following their exposure to hospital personnel harboring these resistant organisms has reached major proportions with an ever-increasing number of deaths [10] being reported from the resulting sepsis.

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The treatment of these patients therefore needs to return once again to the use of antigens capable of stimulating immune body production as well as being effective in desensitization to acquired bacterial sensitivity. In a previous study [11] it was shown that Hoffmann's acellular bacterial antigen complex was effective in a group of 50 patients with recurrent upper respiratory infections and infectious asthma. Shinefield,[12] using autogenous acellular bacterial antigen complex (Hoffmann), obtained similarly good results in the treatment of 110 children suffering from chronic, persistent, and recurrent upper respiratory infections and infectious asthma. Dalven and Romano [13] have confirmed the effectiveness of stock preparations of Hoffmann's acellular bacterial antigen complex in 22 children suffering with these diseases.

The present report concerns the use of Hoffmann's [14] acellular bacterial antigen complex (B. A. C.)* in the treatment of 200 patients with recurrent complicated

* The acellular bacterial antigen complex preparations were obtained through the cooperation of the Hoffmann Laboratory, 666 Broadway, Paterson, N. J.

TABLE 1.—*Selection of Patients.*

TOTAL NUMBER OF PATIENTS TREATED****	200 WITH COMPLICATED COLDS

CLASSIFICATION OF COMPLICATIONS

WITH SINUSITIS----	74
WITH INFECTIOUS ASTHMA-	64
WITH BRONCHITIS-	51
WITH THROAT INFECTIONS	11

TABLE 2.—*Classification of Nonbacterial Sensitivities*

Diagnosis	Positive Family History of Allergy, %	Positive Pollen Sensitivity, %	Positive Dust Sensitivity, %	Positive Inhalant Sensitivity, %	Positive Food Sensitivity, %
Sinusitis	54	25	38	5	4
Infectious asthma	48	30	44	9	1.5
Bronchitis	35	14	22	10	0
Throat infections	45	9	9	9	0

colds followed for a period up to three years. The antigens prepared by the Hoffmann process are complex in nature. The antigenic components of the bacteria and their metabolites are included in this complex preparation in a noncellular readily diffusible state, essentially free of extraneous elements of the culture media such as meat extracts and peptones.

B. A. C. Hoffmann is not a vaccine. The bacteria in vaccines, which are cellular antigens, when injected into the patient must first be lysed in order that their antigenic components may be useful for immune-body production and desensitization. In addition, vaccines, unlike the Hoffmann acellular bacterial antigen complex, lack the soluble specific substances lost in the washing process, and also are lacking in the important bacterial exotoxins and other metabolic products present in the bacterial filtrate only.

Selection of Patients

Patients were selected for treatment (Table 1) only if their history indicated poor bacterial resistance or hypersensitivity to bacteria. This was indicated by the history of perennial, recurrent, severe complicated colds and infectious asthma.

The presence of an acute infection was not considered a contraindication for treatment. Forty-six of the patients had acute infections when first treated, and of these twenty-one had fever.

Classification of Patients Selected

Patients treated were classified (Table 2) according to the type of their bacterial complications, the presence of asthma, the presence of a positive family history of allergy, and the presence of nonbacterial sensitivities.

Of the 200 patients treated, 74 had developed sinusitis, 64 infectious asthma, 51 bronchitis, and 11 throat infections.

Of the sinusitis patients 54% gave a family history positive for allergy; the same was true for 48% with infectious asthma, for 35% with bronchitis, and for 45% with throat infections.

About 30% of the patients with sinusitis and infectious asthma were found sensitive to pollens and about 40% sensitive to dust. Patients with bronchitis and throat infections showed one-half or less the amount of sensitivity to pollens and dust. Other inhalant and food sensitivities were found in only 10% of the total number of patients treated.

Forty-one patients, or approximately 20%, were allowed to stop treatment for periods of 6 to 12 months in order to observe differences in results during the treated and untreated periods.

Materials

B. A. C. Hoffmann, a water-clear solution, is the product of a special exclusive method whereby the soluble specific substances obtained from bacteria such as the "M" substance in group A hemolytic streptococci, the polysaccharides in pneumococci, or the Boivin antigens of Gram-negative organisms, are combined with the respective toxins and enzymes produced by pathogens in vitro and vivo.

The specific B. A. C. Hoffmann preparations used (Table 3) were as follows:

1. Respiratory B. A. C. prepared from virulent cultures of streptococci (hemolytic, nonhemolytic, and viridans type), pneumococci, staphylococci, Bacterium coli, Neisseria catarrhalis, and Hemophilus influenzae isolated from active infections and stock strains of nonhemolytic streptococci and hemolytic streptococci, Types 2, 3, and 12.

TABLE 3.—*Types of Bacterial Antigen Complex Used for Treatment*

Diagnosis	Sex		Children			Adults		
	M	F	No.	Respiratory B. A. C.	Pooled Group B. A. C.	No.	Respiratory B. A. C.	Pooled Group B. A. C.
Sinusitis	32	42	14	13	1	60	39	21
Infectious asthma	33	31	27	17	10	37	20	17
Bronchitis	24	27	10	7	3	41	33	8
Throat infections	4	7	4	1	3	7	7	0
Totals	93	107	55	38	17	145	99	46

2. Gram-negative B. A. C. prepared from cultures of Bacillus pyocyaneus (Pseudomonas aeruginosa), Escheric coli, and Bacillus lactis aerogenes.

3. Pooled group strains B. A. C. prepared from typed strains of eight selected groups of bacteria listed below:

Beta hemolytic streptococci, Group A, Vial 1, Griffith Types 10, 14, and 19; Vial 2, Griffith Types 3, 12, and 18

Pneumococcal types (most prevalent in adults), Vial 3, Diplococcus pneumoniae Types 1, 2, and 3; Vial 4, Diplococcus pneumoniae, 5, 7, and Klebsiella pneumoniae (Friedländer's bacillus)

Pneumococcal types (most prevalent in children), Vial 5, Diplococcus pneumoniae Types 1, 6, and 14; Vial 6, Diplococcus pneumoniae, Types 5, 7, and 19

Streptococcus, viridans type, Vial 7 Strains R 1551, R 1822, and R 353

Various organisms Vial 8, Staphylococcus aureus and albus, Neisseria catarrhalis, Hemophilus influenzae A and B, and B. coli

All B. A. C. Hoffmann preparations used were standardized to contain 0.120 mg. of total nitrogen per cubic centimeter.

Method

All inoculations were given intracutaneously, using a 3/8 in. 27-gauge needle and a tuberculin syringe. The inoculations were given into the skin of the middle of the arm and forearm. No more than two preparations were given on the same visit. All sites were at least 1½ in. apart.

The initial dose consisted of 0.02 cc. and this same dose was maintained throughout the period of treatment. The inoculations were given once weekly for four weeks, at two-week intervals for another four weeks, and then once monthly thereafter.

Treatment was given during the presence of an acute infection as well as between infections. All patients received treatment with the Gram-negative B. A. C. in addition to the other selected B. A. C. preparations. This was carried out because the frequency of previous antibiotic therapy in this group of patients being extremely high, it followed that in many the Gram-negative organisms had been allowed to multiply and become pathogenic. All patients found sensitive to pollens and dust received injections of these while under treatment with acellular B. A. C.

One hundred thirty-seven patients were treated with respiratory B. A. C. Hoffmann. Sixty-three patients were treated with pooled group strains B. A. C. Hoffmann. Selection of the particular pooled group strains used depended on the patients clinical response. Only clinical response was used as an indication for treatment with a particular preparation. After the maintenance of improvement each patient on these pooled group strains was given an inoculation of the all-inclusive respiratory B. A. C. Hoffmann preparation. This was then repeated at intervals throughout the treatment of these patients.

Skin Tests

Since all inoculations were given intradermally, observations of skin-test reactions were easily made (Table 4). Immediate wheal type skin-test reactions were observed in 65% of the patients treated with Hoffmann B. A. C. Correlation between the

TABLE 4.—*Results of Skin Tests, Immediate Type*

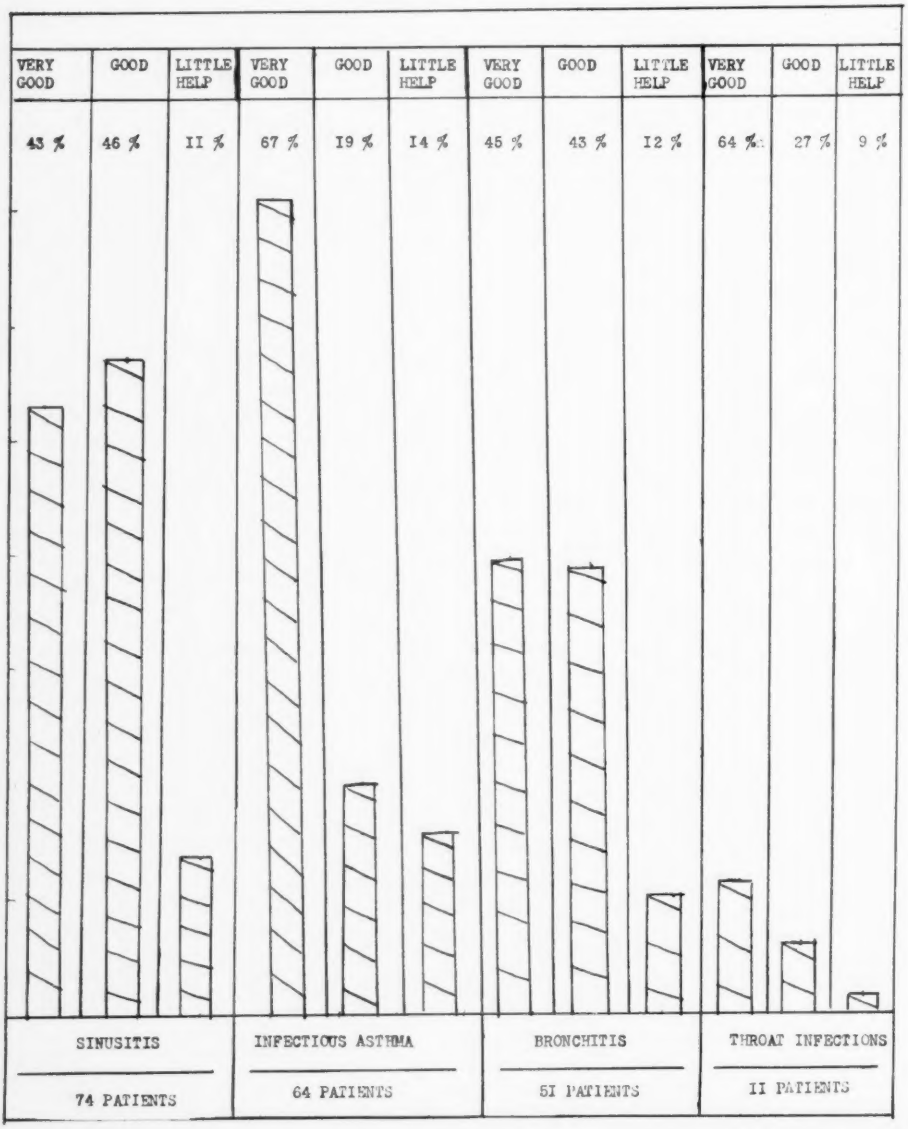
Diagnosis	Type of B. A. C. Hoffmann Used for Skin Test					
	Respiratory B. A. C. Vial		Pooled Group B. A. C. Vials		Total Positive, %	
	No. of Children Positive	No. of Adults Positive	No. of Children Positive	No. of Adults Positive	Children	Adults
Sinusitis	7	27	1	12	57	65
Infectious asthma	12	5	8	15	74	54
Bronchitis	5	16	1	7	60	56
Throat infections	0	4	3	0	75	57

skin-test reaction and the clinical response to treatment was not observed consistently.

More positive skin tests were obtained with the B. A. C. preparations made from pooled, stock typed specific bacteria used in a separate set of eight vials than with the single vial of respiratory B. A. C. containing the entire number of groups of specific bacteria.

Very likely the immediate wheal-type skin test observed with acellular bacterial antigen complex preparations is due to the presence in them of soluble specific bacterial polysaccharides and proteins as well as numer-

TABLE 5.—*Results of Treatment with Bacterial Antigen Complex Hoffmann in 200 Patients with Complicated Colds.*



ous toxins and enzymes produced by pathogens in vitro and vivo.

When patients were tested with the individual set of eight vials, it was found that the vial containing S. aureus and albus, N. catarrhalis, H. influenzae Types A and B, and B. coli, gave the greatest number of positive skin-test reactions. A biopsy of a skin site tested with acellular bacterial antigen complex reveals almost no cellular reaction. This is in marked contrast to a biopsy of a skin site tested with a vaccine; here one finds an immense cellular reaction with infiltration into the area of large numbers of leukocytes. The vaccine, being a cellular preparation, attracts the leukocytes whose aim is to engulf the bacteria and destroy them. This sets up an inflammatory focus, which so frequently is noted 24 hours later as a so-called delayed positive skin test reaction.

Observations of repeated skin tests with acellular bacterial antigen complex revealed the following: In some patients, as is seen with pollens and other inhalants, the skin test remained positive. In some patients, as is seen with diphtheria toxin-antitoxin, the skin test became entirely negative, and in some it became reduced in size.

The disappearance of the positive skin test was taken as evidence that it originally was due to one of the bacterial toxins. The development of such a negative skin test was therefore probably due to the production of antitoxin in the patient tested. For the same reason it was considered very likely that in those patients whose skin tests decreased in size only part of the positive test was due to toxin sensitivity.

Preliminary observations on patients whose skin tests remained positive have shown that it is possible to get a positive passive transfer test with use of their serum. This would indicate the presence in these patients of reagins which are able permanently to react with acellular bacterial antigen complex. Results of further studies with passive transfer testing will be reported in another paper.

A number of patients in this study have shown an initial and persistently negative skin test. Experience has shown that these patients require the least number of injections, and many of them remain well on four to six treatments a year with Hoffmann's B. A. C. These patients very likely are not hypersensitive to bacteria but need instead only to be better immunized.

Results of Treatment

The 200 patients treated in this study all had one thing in common, a history of repeated complicated colds. Treatment with the new acellular bacterial antigen complex (Hoffmann B. A. C.) brought about a remarkable reduction in the frequency of these infections (Table 5).

Results were considered very good if the patient no longer was troubled with complicated colds. With such improvement a patient might still develop a cold, but such a cold would now run a typical course and would no longer cause the complications of sinusitis, pharyngitis, laryngitis, tracheitis, bronchitis, bronchiectasis, and infectious asthma.

Results were considered good if the patient had a marked reduction in the number of complicated colds and in the severity of the complications when they occurred.

Results were considered a failure if a marked reduction in the number of complicated colds did not occur.

Of the 200 patients treated, 105 showed very good results, 71 good results, and 24 were considered unimproved. Improvement with reduction in the incidence of repeated complicated colds usually started soon after therapy with the new acellular bacterial antigen complex was begun and was followed by a similar improvement in the chronic sequelae involving the sinuses and lungs. Chronic nasal discharge lessened markedly; bronchial coughs ceased, and patients with infectious asthma soon noted a lessening and disappearing of their attacks.

The effectiveness of these bacterial antigens was clearly demonstrated in the control

group of 41 patients. In this group, during the 6- to 12-month period of omission of such treatment, there was a gradual increase in the frequency of complicated colds previously noted. In addition, the secondary effects of these complicated colds—sinusitis, bronchitis, and infectious asthma—again appeared. Following a return to treatment with the acellular bacterial antigen complex improvement was again noted in this group. There was again a marked reduction in the frequency of complicated colds and a marked lessening of the postnasal discharge, bronchial cough, and infectious asthma.

The period of observation covered in this study extended for three years in 31 patients, for two years in 60 patients, for 18 months in 53 patients, and for one year in 56 patients. During this one- to three-year period of observation the good results obtained with the new acellular bacterial antigen complex has been maintained in 88% of the patients under such treatment.

Summary and Conclusions

The common cold is our greatest single cause of illness. Although the common cold has a specific viral etiology, its most frequent complications are bacterial. Such complications frequently start in early infancy and remain a burden to the patient for many years. Of the 200 patients selected for treatment in this study, 74 had developed sinusitis, 64 infectious asthma, 51 bronchitis, and 11 throat infections.

Antibiotic therapy has not solved the problem of recurrent upper respiratory infections. The indiscriminate use of antibiotics has interfered with immune-body production and has been of no value in producing desensitization to bacteria or their products. Such therapy, furthermore, has resulted in the development of resistant strains of bacteria in the overgrowth of a normally depressed bacterial flora, which frequently is equally detrimental to the patient.

The treatment of these patients therefore needs to return once again to the use of antigens capable of stimulating immune-body production as well as being effective in desensitization to acquired bacterial sensitivity. Several previous reports have confirmed the effectiveness of Hoffmann's acellular bacterial antigen complex in the treatment of patients with severe recurrent upper respiratory infections and infectious asthma. This report adds further confirmation of its effectiveness in the treatment of 200 patients with complicated colds. The results were good in 88% of the patients under such treatment followed for periods up to three years.

There were no significant side-effects noted in the 200 patients treated in this study. Treatment was carefully controlled with a fixed dosage of antigen at each visit. No patient received more than two injections of 0.02 cc. of the antigens at each visit; the concentration of the antigens, 0.120 mg. of total nitrogen per cubic centimeter, was kept constant throughout the study.

In 88% of the patients treated with Hoffmann's acellular bacterial antigen complex improvement was noted soon after therapy was begun. Improvement in many patients was promptly followed by a marked reduction of the bacterial complications such as sinusitis, bronchitis, and infectious asthma. Experience has shown that patients with an initial and persistently negative skin test to these antigens require the least number of injections and many of them remain well on four to six treatments a year. These patients presumably are not hypersensitive to bacteria but need instead simply to be immunized with an increase in their resistance to upper respiratory infections.

As is so well discussed by Anderson and Rubin, the "allergic load" is increased with poor nutrition, anemia, and other noninfectious allergic factors. It is therefore emphasized that all patients with repeated infections be given the benefit of a complete medical and allergic study and that treatment be given for all of the conditions found as well as with the acellular bacterial antigen complex preparations (Hoffmann).

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REFERENCES

1. Top, F. H., and others: Communicable Diseases, Ed. 3, St. Louis, The C. V. Mosby Company, 1955, p. 596.

2. Flippin, H. F.: The Common Cold from the Viewpoint of the Internist, Pennsylvania M. J. 58: 29 (Jan.) 1955.

3. Woodward, J. F., Jr.: The Changing Practice of Otolaryngology, A. M. A. Arch. Otolaryng. 64: 486 (Dec.) 1956.

4. Solinger, S.: The Paranasal Sinuses, A. M. A. Arch. Otolaryng. 65:47 (Jan.) 1957.

5. Prigal, S. J.: Intrafamilial Contagion in Chronic Sinorespiratoryi Infections, Dis. Chest 25: 448 (April) 1954.

6. Carpenter, P. L.: Immunology and Serology, Philadelphia, W. B. Saunders Company, 1956, p. 225.

7. Christenson, W. N.; Hedrich, G. W., and Schugmann, R. F.: Fatal Anaphylactic Reaction Following Penicillin Injection, U. S. Armed Forces M. J. 4:249 (Feb.) 1953.

8. Dowling, H. F.; Lepper, M. H., and Jackson, G. C.: Clinical Significance of Antibiotic Resistant Bacteria, J. A. M. A. 157:327 (Jan. 22) 1955.

9. Loh, W. P., and Abiog, A.: Staphylococci in a Community Hospital: II. Nasal Carrier Rates of Newborn Babies and Their Mothers, New England J. Med. 256:179 (Jan. 24) 1957.

10. Levin, M. N.: Staphylococcal Hospital Infectious, New England J. Med. 256:155 (Jan. 24) 1957.

11. Spielman, A. D.: Treatment of Infectious Asthma and Respiratory Infections with an Acellular Bacterial Antigen Complex, New York J. Med. 55:1603 (June 1) 1955.

12. Shinefield, M. A.: The Use of Autogenous Acellular Bacterial Antigen Complex in the Treatment of Recurrent Respiratory Infections and Asthma in Infants and Children, New York J. Med. 56:1466 (May 1) 1956.

13. Dalven, J., and Romano, F. J.: Pilot Study Impressions on Treatment of Infectious Asthma and Respiratory Infections with Acellular Bacterial Antigen Complex in 22 Children, New York J. Med. 57:1748 (May 15) 1957.

14. Hoffmann, J.: Method presented at the 41st Meeting of the New York City Branch of the Society of American Bacteriologists, Oct. 28, 1947.

The Adenotonsil Problem

A Review of Five Thousand Cases

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It is a pleasure to present this so-called controversial subject, the adenotonsil problem. One could aptly apply this to the story which Bill Cunningham, eminent Mutual Commentator of Boston told a few months ago.

There was a man who was quite ill and apparently approaching the time when he might pass into the great beyond. The preacher went out to see him, and said to him: "Have you renounced the devil?" The reply from the man was: "No Sir, I haven't, and considering my present condition and the type of life that I have lived, at this moment I just don't want to antagonize anyone!"

So I do not wish to antagonize anyone in my comments upon the adeno-tonsil problem. If you will read the articles in the Feb. 16, 1957, *Journal of the American Medical Association* by Drs. Galloway, Baron, and Weiss, you will have a rather comprehensive review of this subject.

Shakespeare in his writing has Antony eulogizing over the body of Caesar and he states something as follows: "The noble Brutus has told you that Caesar was ambitious; if it were so it were a grievous fault and grievously has Caesar paid for it. Caesar brought many captives home to Rome whose ransom did the general coffers fill." I have brought many tonsils and adenoids home, whose ransom has my coffers filled. Sons and daughters have been sent to school from the lucrative gain from adenotonsillectomies. Almost every living physician or surgeon, with few exceptions, has at some time in his training, internship, residency, or at various and diverse places performed an adenotonsillectomy. One could almost weep over the atrocities that have been performed in the throat, and yet with due respect we approach with reverence pertinent germane thoughts concerning the same.

Tonsillectomies are not cure-alls. One cannot anticipate doing something phenomenal to certain patients by doing adenotonsillectomies or a tonsillectomy.

It is necessary in the evaluation of the adenotonsillectomy problem to assume an unbiased opinion. No dogmatic edict is applicable to every case so keen rationalization must be employed in conceiving an answer.

Tonsil Structure: Buccopharyngeal mucosa with lymphoid follicles, covered with epithelium and a chorion filled with lymphoid formations. Epithelium of Malpighian type, penetrates depth of the organ forming crypts or sulci. Epithelium is of stratified squamous cell type.

Pharyngeal Lymphatic Ring of Waldeyer

1. Pharyngeal bands.
2. Pharyngeal granulations.
3. Lingual tonsils.
4. Palatine tonsils.
5. Adenoids.
6. Tissue in Rosenmüller's fossa.
7. Tubal tonsils.

Blood Supply

1. Dorsalis linguae from the lingual.
2. Ascending palatine and tonsillar from the external maxillary.
3. Ascending pharyngeal from the external carotid.
4. Descending palatine branch of the internal maxillary.
5. Twig from the small meningeal.

Nerves

1. Middle and posterior palatine branches of the maxillary.
2. Glossopharyngeal.

Alfaro has very aptly stated regarding the functions of the tonsils and adenoids:

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1. Tonsils and adenoids constitute a barrier to respiratory infection in infancy and childhood.
2. This barrier is a site for the development of antibodies which will protect the child in future years.

Boies argues for the validity of these functions in spite of the fact that they are so frequently lost in childhood as a result of infection and hypertrophy; he thinks that their purpose could still have been fulfilled in the age period when the need may be the greatest.

Indications for Adenotonsillectomy

1. Repeated attacks of tonsillitis with an accompanying adenoiditis and usually complicated by regional cellulitis, recurrent adenopathy with hyperpyrexia, general malaise, and bizarre pains which could be indicative of early rheumatic symptoms.
2. Abnormal hypertrophy of the adenoid and tonsil tissue to the extent of definite obstruction to nasal ventilation, impaired deglutition, and improper and inadequate growth, not only of regional structures but general systemic debility.
3. Otitis media—which may be involved in the above-mentioned obstructive process together with an infectious tubal catarrh. Recurrent otitis media catarrhal or purulenta are serious, and every measure must be considered to cope with them. Deafness following repeated bouts of adenoiditis is too significant to pass by lightly. Personally in these cases I cannot recommend simple adenoidectomy—do an adenotonsillectomy!
4. Chronic tonsillitis and adenoiditis, contributing factors in chronic sinusitis, chiefly from an obstructive standpoint (adenoids). Allergy may enter the picture and challenges our diagnostic acumen. Allergic and infectious rhinorrhea, differentially diagnosed by examining a smear of the nasal secretion with Hansel's stain for eosinophil count.
5. Focal infection and systemic disease have ardent supporters in the pro and con column. Statistics are quoted in their arguments.
6. Vincent's tonsillitis, peritonsillar and intratonsillar abscesses, diphtheritic tonsillar infection (carrier factor) are indications for tonsillectomy.

Contraindications for Adenotonsillectomy

1. Never operate during an epidemic of severe proportions, be it measles, mumps, poliomyelitis, et cetera.
2. Never operate during an acute infection because the already inflamed tissue does not react kindly and inciting a severer process, laryngospasm, bronchospasm, or respiratory failure may occur.
3. Never do a "hopeful tonsillectomy" without precisely explaining to the parents or the older patients that this is an elective procedure and being hopeful that this will cure any and all complaints is too much to expect.
4. Neuroses, psychic trauma, fear, misgivings, et cetera.
5. Blood dyscrasia, hemophilia, anemia, et cetera.
6. Menstrual cycle.
7. Anomalous internal carotid.

Reiterating the question concerning poliomyelitis and tonsillectomies, with the advent of the Salk vaccine this problem will eventually be solved. We must emphasize, however, never do any elective surgery during any epidemic regardless of nature.

Therapy

X-ray and radium, except in very rare circumstances, cannot be a commendable therapeutic measure. It is possible that under certain exceptional circumstances where the Eustachian tube orifice has lymphoid tissue in it that it can be a recommended procedure; otherwise, it should be deleted from the armamentarium of adenotonsil therapy. Nor can I in any way condone electrosurgery (cautery) at weekly sittings attempting to desiccate the tonsil tissue. I have, in days gone by, removed the remnants of cases who have in other hands had electrosurgery desiccation of the tonsils. I have personally never seen a complete removal of tonsils by this procedure, nor have I ever used this procedure. Careful dissection with complete enucleation as discussed under Technique is the procedure of choice.

Preoperative Thinking

1. Be sincere in discussion of the adenotonsillectomy to the parents and the child. Full cooperation is desired. Cahn has recommended that the small booklet by J. A. Sever, "Johnny Goes To The Hospital," be made available to every parent so that proper indoctrination may be accomplished before the day of the hospitalization.

2. Routine penicillin preoperatively, during, or immediately after surgery should be

considered but not universally given unless definitely indicated. Streptococcus of the viridans type, previously diagnosed, may cause a bacteremia with severe systemic reaction; therefore, adequate protection is necessary. Sulfonamides and other antibiotics pre- and postoperatively may be justified.

3. Sedation before surgery must be judiciously used; unwisely used it may be extremely dangerous. Atropine or scopolamine (hyoscine) usually reduces to a minimum the vasovagal reflex and reduces secretions.

4. Bleeding and clotting time and general physical examination. Blood dyscrasias and anemias may be found. Hemophilia can be ascertained usually by interrogating the family. If the threshold of the bleeding and clotting time are abnormal, we use conjugated estrogen substances (Premarin) both intravenously and orally as necessary, and reevaluate the findings before surgery.

Adenotonsillectomies are not simple operative procedures. Never undertake this surgery unless you are thoroughly familiar with and able to master every complication that may enter the scene.

Complications

1. Cardiac arrest.
2. Hemorrhage, immediate or delayed.
3. Anatomical anomalies, abnormal styloid process, vascular aberrations.
4. Anesthetic problems.
5. Subcutaneous emphysema.
6. Tracheobronchial blood clots from hemorrhage.

Technique

Careful dissection and complete enucleation of the tonsil should be carried out according to your own choice, but make it simple and complete. Visualize directly the adenoid area before completing the surgical procedure. Never allow a patient to leave the operating room until any and all bleeding is controlled. Endotracheal anesthesia has been highly recommended by Pender and Hallberg in reducing hazards in adenotonsillectomies.

Post-Tonsil Syndrome. This consists of pain in the throat and neck, lump in the throat, swallowing difficulty, and breathing difficulty. This may last for weeks, months, years, and is usually due to incarceration of the nerve tip endings and/or mild contraction of the palatoglossus and/or palatopharyngeus muscles following surgery.

Summary and Conclusion

During the past quarter of a century over 5000 tonsillectomies or adenotonsillectomies have been performed. The results have been gratifying and the constellation looks brilliant, but there are a few shadows. It takes courage to report one death, one near death with aspiration of blood from the tracheobronchial tree; one uvulectomy; 5 post-tonsil hemorrhages of severe proportions; 50 to 100 additional hemorrhages of various assortments. One patient contracted poliomyelitis a few days after adenotonsillectomy but lived and suffered no residua from the same; several whom I know developed tracheobronchitis; and possibly one or two had severer complications (bronchiectasis questionable).

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Postoperative Hemorrhages Related to Atmospheric Changes

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The significance of the relationship between weather changes and the human body was well known 25 centuries ago and was discussed by Hippocrates, the world's first meteorologist and the father of medicine. Numerous studies and publications discussed the effect of atmospheric changes upon the different organs and systems of the body. In otolaryngic literature, Fabricant pointed out the correlation between the weather influences and incidence of acute mastoiditis and acute tonsillitis. Petersen dealt with the biochemical and vasomotor changes occurring in men during atmospheric alterations. Boles reported at the annual meeting of the American Medical Association in 1954 on the increased incidence of duodenal and gastric ulcer hemorrhages in certain months. He felt that barometric pressure and normal temperature changes may be correlated with the incidence of hemorrhage in duodenal ulcer. In his opinion, temperature variations put an added stress on the body which results in bleeding. Others report about a sudden apparent onset of or an increase in severity of eclampsia during the sudden change of weather. It is well known that resistance to bacterial infections lessens during sudden temperature fluctuations. During a thunderstorm adults and children become more restless and irritable.

The topic of this discussion is the observation pertaining to primary and secondary post-tonsillectomy and adenoidectomy hemorrhages coinciding with atmospheric changes on certain days. In personal communication, numerous colleagues performing adenotonsillectomies admit the likelihood of such relation in postoperative bleedings and confirm similar experiences.

I could not find any publication in contemporary literature on this particular subject, except the report of Merten of 1942, from the Vienna Otolaryngological Society, describing secondary post-tonsillectomy diffuse tissue bleedings (capillary hemorrhages), on four patients on one rainy and damp afternoon. There was no apparent cause or explanation for the bleedings, and the Vienna Institute for Meteorology and Geodynamics confirmed the marked changes and fluctuations of the air pressure during the same afternoon. His statistical studies proved higher incidence of bleedings when weather fronts passed over the area.

My experience is based on five years' observation, carefully considering all factors which may possibly be involved, in primary and secondary hemorrhage after tonsil and adenoid surgery. Among the potential complications of this operation, bleeding is the commonest. Tonsillectomies constitute approximately one-third of all surgical interventions of our country. The procedure should not be considered as minor surgery for many reasons. Prevention or minimizing of the hemorrhage is one of our most important requirements for the smooth postoperative course and full surgical success.

The total percentage of bleedings in this survey corresponds with the average values. The atmospheric conditions during the hours of incidence of hemorrhages were carefully studied in cooperation with the Meteorologist and State Climatologist of the U. S. Weather Bureau of Columbus, Ohio,

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using their official records. Also postadenotonsillectomy bleedings other than my cases, occurring at the Chillicothe Hospital, were evaluated. The history and character of a total of 53 cases of postoperative hemorrhage were analyzed. They occurred in 1721 tonsilloadenoidectomies as primary or late primary hemorrhages within five hours after surgery. No prevalence was noted in different ages or sex. The remarkable observation was that the time of the majority (57%) of primary hemorrhages coincided with the "cold front" and usually every winter with the snowfall. Two or three bleedings occurred on such days. Similar situations appeared on days with fluctuating barometric pressure (well marked atmospheric variations) or on days with fluctuation of the relative humidity (83%-59%-91%) without pressure changes. There was more bleeding during the surgery and prolonged capillary oozing postoperatively. In other cases rapid pressure changes with thunderstorms coincide with the time of bleeding. General surgeons frequently experience capillary oozing and increased tissue bleedings on such days.

It is not my intention to state that atmospheric changes are the most important factor in bleeding. There are many other and well-known factors. It has been repeatedly proved and published in recent years by American and European writers that weather changes are associated with vascular constriction followed by dilatation, with changes in blood-sugar level, basal metabolic rate, blood pressure, capillary permeability, etc. It should be emphasized that weather is only one of the factors influencing the individual's responses. It is of the utmost importance in statistical surveys and in our studies to rule out the coincidental occurrence of bleeding from other cause during the atmospheric alteration. Objective facts confirm that there is a certain relation, perhaps only in some individuals, with other predisposing factors. Some persons are less capable of satisfactory adjustment to atmospheric changes than others. If the cold front may precipitate acute tonsillitis, common colds, etc., the hemorrhage could easily be related to vascular changes, as vasodilation and hyperemia, in the area of pharynx. According to Fabricant's studies, changes are described in functional status of the nasal and pharyngeal mucous membranes. Under these circumstances perhaps the posttonsillectomy bleeder was a cold carrier in early latent incubation period or "lag" during the surgery, owing to the atmospheric conditions, and the early postoperative oozing was the first clinical manifestation of the acquired infection. During this period of the changed functional status of the nasal and pharyngeal structures the capillary permeability is also increased. On the other hand, the maladjustment to in- and outdoor temperature changes could be due to the chilling effect in an over-airconditioned operating room on a susceptible person.

Petersen pointed out that each passing atmospheric alteration is associated with changes of all the physiological processes, because each change in the air mass requires prompt autonomic adaptation. Meteorological alterations are associated with corresponding changes in blood and tissue chemistry, with changes in autonomic (vasomotor) and endocrine systems. High and abrupt barometric fluctuations cause profound changes in functional capacity of mucous membranes and adjacent structures.

Tintera and Goldman assumed that oozing after tonsillectomy is due to capillary fragility from endocrine disturbances. If so, the primary factor could be the atmospheric condition in certain cases, according to Petersen's studies. During the passage of polar air mass, the capillaries are less permeable; blood pressure is increased; the blood becomes more alkaline (pH increased). Gradually the vessels become dilated to accommodate the increased demand of the organism for greater O_2 consumption; the blood pressure falls; the membranes become more permeable, and the tissues become relatively more acid. The forces mentioned influence the metabolic

processes, causing a shift of organic mechanism over the edge of normality (Fabricant).

Curry, an authority in the field of aerodynamics, demonstrated that ARAN (one of the oxygen polymers in the atmosphere) may be the direct factor in altering the susceptible human organism. The ARAN content in the air mass is increased during and after an electromagnetic disturbance, which is responsible for the autonomic disintegration of the sensitive individual.

Curry, in his study of bioclimatics differentiates three types of men as far as weather sensitivity is concerned: (1) W-type, sensitive to warm front. (2) K-type, sensitive to cold front. (3) G-type, indifferent to weather changes. According to this classification, types W and K may develop biologic changes with alteration of the capillary bed, predisposing or leading to vascular accidents under the influence of atmospheric conditions.

All students of the weather problem are convinced that sudden storm disturbances and changes of barometric pressure do induce marked disturbances in body functions, resulting in functional disabilities. Peripheral vascular changes due to vasomotor dysfunction, increased capillary permeability, and perhaps fragility due to possible endocrine imbalance are some of the results.

The research of German meteorobiologists Schultze (Hamburg) and Reiter (Munich) leads to conclusions that atmospheric turbulances produce electromagnetic changes, radiation of high frequencies, infralong waves, and disturbances of the air-electrical field. These changes act upon the human body as biotropic factors as well as any artificial or natural waves. Their primary action upon the body colloids results in latent or manifest acidosis. Action through the humoral system upon the vegetative nervous system is secondary or indirect, according to Pfanner. The resulting autonomic disorder could be one of the factors, explaining the peripheral circulatory or capillary changes, causing tissue oozing.

It is emphasized, that these considerations present a preliminary study in correlation of weather changes and vascular pathology in course of tonsil and adenoid surgery. It should serve as a stimulation for more comprehensive work from different areas of the country, to clarify the pathophysiology and pathogenesis of this type of postoperative bleeding, and to establish a possible prevention.

Summary

There is evidence of increased frequency of postoperative bleeding and capillary oozing during atmospheric turbulances, on days with abrupt sudden fall of the barometric pressure, during thunderstorms and barometric fluctuations, and at the onset of cold front (polar mass).

During five years' observation on 1721 adenotonsillectomies, 57% of primary and late primary (within five hours postoperatively) hemorrhages were related to atmospheric changes where no other causes could be proved.

It is presumed that in susceptible persons weather changes may induce generalized body reactions with autonomic disorder which may result in postoperative bleeding.

53 E. Second St.

BIBLIOGRAPHY

Boles, R. S., and Westerman, M. P.: Seasonal Incidence and Precipitating Causes of Hemorrhage from Gastric Ulcer, J. A. M. A. 156: 1379 (Dec. 11) 1954.

Curry, M.: Bioklimatik, Munich, 1946; cited by Petersen and Pfanner.

Fabricant, N. D.: Climatic Factor in Mastoiditis, Arch. Otolaryng. 30:549 (Oct.) 1939.

Climatic Factor in Precipitating Acute Tonsillitis in Children, J. Pediat. 15:697 (Nov.) 1939.

Weather and the Climate, Am. J. M. Sc. 200: 835 (Dec.) 1940.

Otolaryngology and the Weather, Illinois M. J. 78:511 (Dec.) 1940.

Weather and Well Being, Today's Health 33:32 (Jan.) 1955.

Lederer, F. L.: Diseases of the Ear, Nose and Throat, Ed. 6, Philadelphia, F. A. Davis Company, 1952.

Merten, A.: Nachblutungen nach Tonsillektomien, Monatsschr. Ohrenh. June, 1942.

Petersen, W. F.: Man—Weather—Sun, Springfield, Ill., Charles C Thomas, Publisher, 1947.

———and Milliken, M. E.: The Patient and the Weather, Ann Arbor, Mich., Edwards Brothers, 1934.

Pfanner, W.: Föhn und Mensch, Materia Therapeutica, 3-8 (Jan.) Vienna, 1955.

Reiter, cited by Pfanner.

Schütz, W.: Der Einfluss des Wetters auf Erkaltungskrankheiten, gezeigt an Erkrankungen des Halses und der Ohren, Leipzig, Georg Thieme Verlag, 1943.

Schultze, cited by Pfanner.

A Short Study of Papilloma of the Larynx

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In medical literature it is often easier to find information concerning an excessively rare condition than to learn about those which occur frequently. Polyps of the larynx and vocal cords are probably just such a condition.

New growths of the larynx are, unfortunately for mankind in general, common enough; but the innocent or benign wart-like condition known as papilloma is not considered of great importance. Who of the medical profession in general and the lay public in particular think of such "trifling" matter with much concern?

However, we do find in the literature of the world enough information and intelligent knowledge about papilloma of the larynx that would seem to give any experienced laryngologist who is skilled in peroral endoscopy and laryngeal surgery the greatest opportunity to cope with such conditions. The same may be said about all benign neoplasms of the larynx and air passages.

With reference to the general anatomic-pathologic description of papilloma, Jonathan Wright [1] stated many years ago, and it is correct as of today, that the most frequent and characteristic type of papilloma in the larynx is that which to the naked eye resembles a cutaneous seed wart.

The growth as a whole is of rather firm consistency although the surface is soft. Generally speaking, there are, of course types of oval or semiglobular form, but they may be irregular, even angular in contour. In size the neoplasms vary from 1 to 10 mm. or more in long diameter. The color grades from pale gray to pink, and they may be single or multiple.

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It is of great interest that the important observations of laryngologists of yesteryears summarized in this paper are being confirmed. In their pioneer work in connection with indirect or mirror laryngoscopy, they attempted in every way possible to demonstrate the correct observations, which are, perhaps, the observations of most laryngologists throughout the world today.

The site of the laryngeal papilloma is almost always the vocal cord, usually at or near the anterior third or at the anterior commissure; they may appear from any portion of the larynx. On the other hand, tuberculous papillomata frequently grow at the posterior commissure.

From a pathological standpoint papilloma may be considered to possess in its principal elements a connective tissue stroma, hypertrophied papillae, and proliferated epithelium. The stroma may be either soft or compact; the hypertrophied papillae are generally few and simple, each having at the base a central core and covered by layers of epithelial cells, but, generally speaking, multiple and branched. The epithelium may be thick or thin and of the stratified variety. It grows upon, but not at all, as in cancer, into the underlying tissue.

Certain types of papilloma can hardly be distinguished from fibroma except by microscopic study.

From the embryological standpoint, the respiratory passages being derived from the epiblast, its papillomata are considered in the same class as skin warts of the hard type.

Little is known of the etiology of this and the other benign growths of the larynx. A certain tendency or predisposition is evidenced by such neoplasms appearing on various surfaces of the skin, as the neck,

axilla, etc., seemingly related to various inflammatory conditions of a chronic nature.

In view of this fact, some laryngologists regard chronic inflammation as an etiological factor; some believe in the theory of the inherited family tendency. The same etiological factors may be applied to papilloma of the larynx. Laryngeal papillomas have a tendency to recurrence and often require repeated surgical removal; consequently they must be regarded as a surgical disease.

Papillomatosis of the larynx in children [2] is often difficult to treat. The growths are numerous, spread over the vocal cords and larynx and may cause death from suffocation; repeated surgical procedures may be necessary.

Laryngeal papilloma of the vocal cords is the commonest lesion among adults, and the prognosis is quite favorable.

In a most recent report on the subject of managing laryngeal papilloma in children, Kohlmoos [3] discusses the currently accepted method of treatment, which is not generally or universally successful.

Moreover, neoplastic growth frequently subsides before the child reaches the age of 10 years. Excision of papillomata from the larynx at intervals to permit a proper air passage is the best method of treatment. Extralaryngeal surgical procedures, caustics, electrocautery, and x-ray therapy are discounted because of probable injury to adjacent tissue.

Mention should be made of the basic points which undoubtedly are known to all laryngologists but are worth repeating in connection with prognosis and complications. If treatment is careful, the voice should return to normal as the condition regresses. In fact, aphonia may persist if treatment is too harsh.

Generally speaking, the prognosis is influenced by favorable or unfavorable occurrences in conditions pertaining to children.

Some of the earlier authors [4] who collected statistics and studied the prognosis of 127 children with laryngeal papilloma of whom 48 were not operated upon, claimed that of these, 32 died by suffocation. Other authors emphasized tracheotomy as a preliminary measure. These facts probably hold true today.

In fact, a mere glance at the reports in the literature on the subject over the last 10-year period attests to the manner in which the management of papilloma of the larynx has been considered.

For instance, Björk and Weber [5] report studied observations in 117 patients with papilloma of the larynx between 1930 and 1955 (malignant cases were excluded). Of these 71 were male and 46 female. Male predominance was an outstanding fact. They believe that more patients with laryngeal papilloma were admitted to hospitals during World War II and in the postwar period than before the war.

The incidence was increased only in children and young adults and was attributable to difficult social and hygienic conditions. Malignant degeneration occurred in 2 children of a group of 37.

Endoscopic excision of the papilloma was performed in 79 patients, some remaining failures.

Obstruction of the larynx is the most serious complication of untreated papilloma, and lower respiratory obstruction may occur in spite of tracheotomy because tracheal or bronchial implants are not uncommon. Implants may be spontaneous or due to surgical trauma. Stenosis of the larynx and cartilage damage have been observed after radiation or therapeutic laryngofissure operation. Pneumonia and other complications from various causes may result.

In spite of the above frightening complications,[6] prognosis is good if adequate facilities for treatment are available (nursing, assistants, instrumentation, etc.).

There are several factors which influence the progress and prognosis of complications arising from laryngeal surgery; one of them is the method of anesthesia. Therefore, in summarizing the problem of anesthesia I acknowledge my indebtedness to

several of the older observers as well as my contemporaries.

Thus we are reminded in the literature and from personal experience no anesthetic, either local or general, is needed in children. The satisfactory management of direct laryngoscopy without anesthesia in children in connection with laryngeal papilloma has been in vogue in the principal American clinics and hospitals for many years.

Local anesthetic with or without premedication, is used in adults in most cases for direct laryngoscopy as well as laryngeal papilloma. The various kinds of inhalation anesthetics [6] are inconvenient and many of them dangerous because they are explosive.

It is now generally accepted by most of the profession that careful and intelligent planning in connection with personnel or teamwork in the operating room will enable the surgeon to perform perfectly satisfactory endoscopy and laryngeal procedure.

Cases of threatened asphyxia need only prompt and proper management as to instrumentation and methods known to medical science.

The anterior commissure electric laryngoscope is the instrument par excellence for an operative procedure. Correct choice of cupped forceps as to size and type is most important.

432 Surf St.

REFERENCES

1. Wright, J., and Smith, H.: A Text-Book of the Diseases of the Nose and Throat, Philadelphia, Lea & Febiger, 1914.

2. Harris, H. H.: Benign Lesions of the Vocal Cords, Modern Medicine, Vol. 16, Oct. 15, 1948.

3. Kohlmoos, H. W.: Papilloma of the Larynx in Children, A. M. A. Arch. Otolaryng. 62:242-252 (Aug.) 1955.

4. Ballenger, W. L.: Diseases of the Nose, Throat and Ear, Ed. 9, Philadelphia, Lea & Febiger, 1947.

5. Björk, H., and Weber, C.: Papilloma of the Larynx, Acta oto-laryng. 46:499-516 (Nov.-Dec.) 1956.

6. Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat and Ear, Including Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Company, 1945.

Case Reports

Papilloma of the Larynx

A Case of Protracted Duration, 1878-1955

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Papilloma of the larynx appearing in childhood is reputed to have a tendency to be self-limited and to undergo spontaneous regression before or during adolescence. In this respect, it is similar to verruca vulgaris. The viral etiology and contagious nature of the common wart were first demonstrated by Jadassohn [1] in 1895. Ullmann,[2] in 1923, was the first to report similar experiments on the bacteria-free filtrate of macerated papillomatous tissue from the larynx of a child. He produced typical clinical and pathological lesions by inoculating himself and a laboratory assistant with the filtrate. Inoculation with the filtrate from adult papillomatous tissue failed to produce cutaneous lesions. He agreed with Chiari that juvenile and adult papilloma are clinically different diseases.

The precise effect that puberty exerts in the course of juvenile papilloma has never been defined, and it would seem that the theory itself, which has been recently challenged by Björk and Weber,[3] should be revised. The role that viral immunity plays in the spontaneous regression of juvenile papillomata is a subject which should be explored further. It would seem probable that the gross and microscopic tissue changes recognized as squamous papilloma are nonspecific tissue reactions to various irritative agents, one of which is viral.

Some cases of juvenile papillomata may be extremely persistent and may not follow the usual pattern of this disease. I have had under observation five cases of papilloma of the larynx where the disease started in childhood and has continued into adult life. In three of these cases, papillomatous vegetations have been present for 22, 27, and 54 years. One case, which started as juvenile papilloma in a 5-year-old girl, continued and transformed into fatal epidermoid carcinoma. This patient had a laryngectomy at the age of 37 and, in addition to an extensive subglottic carcinoma, had coexisting, benign, squamous papilloma of the epiglottis. The case which I wish to bring to your attention here is one of unusual chronicity and is a progress report on a patient whose illness was first recorded by J. Solis-Cohen [4] in 1893.

Report of Case

The patient was born December 19, 1874, and, after suffering for some time from what was considered to be croup, was seen by Dr. John Atlee of Lancaster, Pa. Dr. Atlee performed a tracheotomy on Sept. 20, 1878, with the patient on the kitchen table of her home in Maytown, Pa. At this time she was almost 4 years of age. Dr. Atlee was certain that the patient had papillomata of the larynx, and four years later, after changing the tube, he planned to hospitalize her for the removal of these obstructing growths. He died, however, before the procedure was accomplished.

The patient was next seen by Dr. J. Solis-Cohen in April, 1892, when she was 17 years of age. She had worn the tracheal cannula continuously for 13

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Dr. Myer Solis-Cohen of Philadelphia and Dr. William A. Atlee of Lancaster, Pa., assisted in obtaining information in regard to this case.

years, and it was badly corroded. She was completely aphonic, and, on laryngoscopic examination, multiple papillomatous neoplasms were seen to fill the upper part of the larynx, laryngeal surface of the epiglottis, and base of the tongue. The growths were removed at multiple sittings during the next month. The exact number of removals is not recorded; however, it is assumed that they were done by indirect laryngoscopic surgery, as this was the custom of the day. After a few weeks, the larynx appeared to be normal, and the tracheal cannula was removed. Four months later, Dr. J. Solis-Cohen performed a plastic closure of the tracheal fistula with a pedicled cutaneous flap. One year later, Oct. 17, 1893, the stoma was closed, and there was considerable improvement in her general health. At this time, however, it was necessary to remove a small papilloma at the anterior commissure.

According to her sister, the patient was a great care to her parents during her early years; however, she went to school and developed normally in every respect. On the playground she was quite a novelty to those with whom she came into contact. She was a good student, and her mother taught her to be a fine seamstress. A few months after the plastic closure (1892), she had a severe case of diphtheria. Upon recovering, she went into nurses' training and later became an operating-room supervisor at the Ithaca City Hospital. In 1907, a nephrectomy was performed by Dr. John Atlee, the grandson of her first doctor. While living in Boston in 1909, age 34, she began to have increasing hoarseness and shortness of breath, and more papillomata were removed, either there or shortly after this when she had moved to Nebraska.

Fig. 1.—Photograph of the patient in the last year of her life (courtesy of Dr. William A. Atlee).

Following this, she had little difficulty, except for a rough voice, for almost 15 years.

She was first seen by my father, Dr. Simon Jesberg, on June 29, 1927, and the record from this date until her death in 1955 is complete. At this first of his many visits, there were multiple papillomatous vegetations of the laryngeal vestibule and epiglottis. The lesions were confined to the area where Dr. Solis-Cohen had encountered them 35 years before. During the next five years, she had 26 direct and 5 indirect laryngoscopic removals of papillomata. The first pathological examination of tissue was on May 16, 1929, and the report was "benign papilloma." At most of the direct laryngoscopies, either the Haslinger directoscope or the Jackson laryngoscope was used. The base was usually fulgurated with electrocautery and, on several occasions, a bismuth solution was injected at the bases of the papillomata. This course of treatment rendered the larynx free of vegetation for varying periods of time, but recurrent lesions always appeared, and the larynx was never free for longer than a period of three months. In spite of the frequent surgical procedures, there was not a great deal of scarring.

The patient was not seen from the middle of 1931 until Sept. 10, 1943, at which time she complained of progressive hoarseness and dyspnea of several years' duration. Examination again revealed multiple papillomatous growths of the laryngeal vestibule. During the next four years, papillomata were removed eight times by direct laryngoscopy.

I first saw the patient Sept. 11, 1947, when she was 72 years of age. There were active papillom-

Fig. 2.—A drawing of the larynx showing multiple papillomatous vegetations of the left ventricular band and the posterior surface of the epiglottis. Anterior and posterior web formations are also present, causing a minor degree of stenosis.

Fig. 3.—A low-power photomicrograph of tissue removed on Feb. 24, 1954, showing the typical appearance of squamous papilloma.

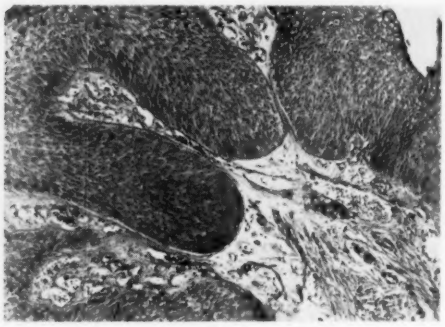

Fig. 4.—A photomicrograph showing typical uniform thickening of the epithelium due to squamous-cell hypertrophy; undulations of epidermal and dermal pegs; and typical stromal changes of increased vascularization, connective tissue hypertrophy, and a small amount of chronic inflammatory infiltration.

atous growths of the left ventricular band and epiglottis, and also small anterior and posterior web formations (Fig. 2). The airway, however, was adequate. During the next seven years, she was under frequent observation and, on four occasions, papillomata were removed by direct laryngoscopy. At times the larynx was free, but never for more than six months.

In 1953, at the age of 78, she began to have cardiac failure, and dyspnea became troublesome. There were some papillomata in the larynx, as usual, and moderate, but not in itself disabling, cicatricial stenosis from web formations, which added to the respiratory embarrassment. It was apparent that a tracheotomy would be necessary, particularly if any further laryngeal surgery was to be performed. Tracheotomy was accordingly advised in March, 1953, but was not performed until Feb. 24, 1954.

At this time, more papillomata were removed, and pathological examination of the tissue was reported as "squamous papilloma" (Figs. 3, 4). On my last examination, April 2, 1954, she was in good condition except for a small amount of papillomatous tissue on the posterior surface of the epiglottis. The tracheal cannula was in place, and she was able to breathe comfortably with it plugged, as long as she was on a maintenance dose of digitalis.

In September, 1954, while visiting her sister in Maytown, Pa., the patient underwent removal of papillomata of the epiglottis by Dr. William A. Atlee, the great-grandson of the doctor who had performed her first tracheotomy in 1878 (Fig. 1). While still in Maytown, she was hospitalized several times for lower respiratory infections. She then had increasing signs of cardiac and renal failure, and died at home on June 11, 1955, in her 81st year. An autopsy was not performed.

Comments and Summary

This patient presents the problem of recurring, benign, squamous papillomata of the larynx for a period of almost 80 years. The care which she required spanned the period of four generations of physicians. I believe that we can say it was chronic. It is interesting to note that the papillomatous vegetations were confined for the most part to the laryngeal vestibule. At no time was there a suggestion of malignant transformation. During the last 28 years of her life, she had 44 laryngoscopic removals of papillomata. The number of times papillomata were removed in the 50 years prior to 1927 is unknown but possibly equals this number.

It may be interesting to reflect on the discoveries which have taken place during the time this patient was under treatment. The incandescent electric lamp was developed by Edison in 1879. The local anesthetic properties of cocaine were discovered in 1880, and its use was introduced into laryngology by Jelinek[5] in 1884. The tubercle bacillus was isolated by Koch in 1882, and the diphtheria bacillus in 1883 and 1884, by Klebs and Loeffler. Pathological examination of biopsy tissue has come into its own, after having received undeserved condemnation in the case of Emperor Frederick III, of Germany, in 1887 and 1888. The fascinating story of this tragedy and the early history of laryngology have been most recently and very interestingly

described by Stevenson.[6] In 1892, the same year that he was treating our patient, J. Solis-Cohen[7] developed the technique of stitching the amputated trachea to the skin of the neck, thus making the operation of laryngectomy more practicable. Roentgen discovered the x-ray in 1895. Jackson,[8] in 1899 and in the years that followed, developed the technique and application of direct laryngoscopy. Wassermann developed his test in 1906. Modern laryngology is built on the foundation provided by these and other more recent discoveries; however, a solution to the problem of recurring papilloma of the larynx still lies ahead of us.

500 S. Lucas Ave. (7).

REFERENCES

1. Jadassohn, J., cited by Ullmann, E. V.[2]

2. Ullmann, E. V.: On the Aetiology of the Laryngeal Papilloma, Acta Oto-laryng. 5:317-334, 1923.

3. Björk, H., and Weber, C.: Papilloma of the Larynx, Acta Oto-laryng. 46:499-516, 1956.

4. Solis-Cohen, J.: Multiple Papillomata in a Child: Tracheotomy at 4 Years of Age; Retention of Canula 13 Years; Evulsion of Multiple Laryngeal and Pharyngeal Neoplasms; Closure of the Tracheal Fistule by a Cutaneous Flap; Recurrent or Repullulant Growth at the Anterior Commissure of the Glottis, M. News 63:664-665, 1893.

5. Jelinek, E.: Das Cocain als Anaestheticum and Analgeticum für den Pharynx und Larynx, Wien med. Wchnschr. 34:1334; 1364-1367, 1884.

6. Stevenson, R. S.: Morell Mackenzie: The Story of a Victorian Tragedy, New York, Henry Schuman, Inc., 1947.

7. Solis-Cohen, J.: Two Cases of Laryngectomy for Adenocarcinoma of the Larynx, New York M. J. 56:533-535, 1892.

8. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939.

A Simplified Technique for Bone Grafting the Eustachian Tube in Radical Mastoidectomy

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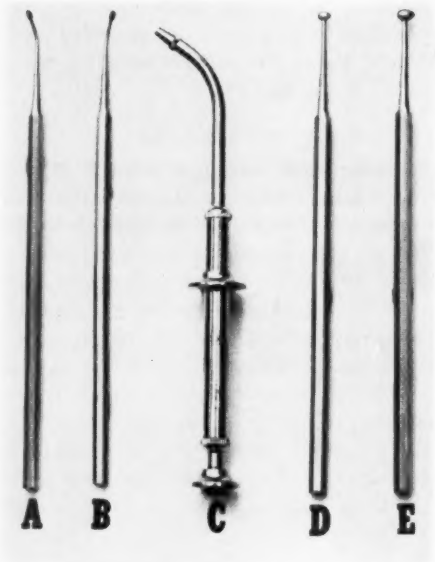
The history of temporal bone surgery, as recorded in the literature, is punctuated with many articles referring to the problem of permanent closure of the Eustachian tube. The number of articles pays tribute to the observation that Kalm[1] made 30 years ago to the effect that "the Eustachian tube is a bugbear. It is almost impossible to close off." Among the many methods advocated, bone grafting is one of the oldest[2] and most widely accepted. During the past three years I have employed a simplified technique for bone grafting, which has to date given very good results.

Technique

The bone dust produced while removing the healthy superficial aspect of the cortex with an electrically driven bur is saved and moistened with enough saline to make thick "mud." The Eustachian tube is carefully curetted and every vestige of mucosa removed. Curved dental scalers (Figure, *A* and *B*) that have a small file on one surface are extremely useful in removing mucosa and providing a fresh bed for the bone graft.* Mucosa in the distal part of the tube which cannot be readily removed is elevated from the bone and pushed down the tube to encourage obliteration. A dental amalgam carrier (Figure, *C*) is a very convenient instrument for the introduction of the bone dust deep into the Eustachian tube. Repeated filling of the carrier with bone mud and packing into the tube will provide a firm plug of bone that fits every contour. The bone mud can be molded and tightly packed with an amalgam burnisher† (Figure, *D* and *E*). A small full-thickness graft taken from the skin of the external auditory canal medial to the isthmus is placed over the powdered bone. Usual packing is employed.



Dental instruments used in the procedure described. *A* and *B*, scalers; *C*, amalgam carrier; *D* and *E*, amalgam burnishers.

Bone dust is always available and obviates the need for additional preparation, drape, incision, and closure. The finely divided nature of this graft is such that vascularization can proceed rapidly, thus minimizing the chance that it will be sequestrated. This method is simple, has a high incidence of good results, and does not increase morbidity.

Results

Thirteen radical mastoidectomies have been done by the technique as described. Whereas the total number of cases is relatively small, there has been only one instance of Eustachian discharge, and all of the ears are dry at this time.

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From The Charlotte Eye, Ear and Throat Hospital.

* Scalers used are Numbers 31 and 32, manufactured by the Starlite Dental Company.

† Amalgam burnishers are Numbers 28 and 29, by the Starlite Dental Company.

Summary

A simplified technique for bone grafting the Eustachian tube has been presented, based on three years' experience with 13 cases.

106 W. 7th St. (2).

REFERENCES

1. Kahn, A.: A New Method for Skingrafting the Mastoid Cavity: A New Method for Closing off the Eustachian Tube at Its Tympanic End in the Radical Mastoid Operation, Laryngoscope 37:889-893, 1927.

2. Ollier, L.: Recherches expérimentales sur la production artificielle des os et sur la régénération des os, J. Physiol. de l'Homme 2:1, 1859; Cited by Adin, L. E., Jr.: The Use of Cancellous Bone Grafting at Tympanomastoidectomy for Permanent Eustachian Tube Closure: A Preliminary Report, Laryngoscope 62:173-193, 1952.

Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Post-Stimulatory Fatigue in Diagnosis

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The classification of hard of hearing persons into conductively and perceptively deaf groups can generally be done with the ordinary pure-tone audiogram when bone-conduction values are related to air-conduction figures. Their difference shows the amount of the conductive component, whereas the gap between bone-conduction thresholds and zero line indicates the perceptive component. However, there are cases in which these criteria do not strictly apply, viz., patients in whom the normal balance between inertia and compression bone conduction is disturbed, or both mechanisms are suppressed. Examples of these are otosclerotic ears with fixed stapedial footplate, and patients with some hindrance in the middle ear limiting the movements of both windows at the same time.[1] In these cases lowered bone-conduction values are found also in conduction deafness.

If the tympanic membrane and the middle ear are otoscopically normal, bone-conduction measurements almost without exception classify the subjects into perceptive and conductive groups. The subdivision of perceptive deafness must be made with other criteria, and for this purpose direct recruitment tests have been employed. The abnormally rapid increase of loudness in recruiting deafness is considered at present to work on the end-organ level. Thus in the presence of adequate numbers of functioning nerve fibers, the damage in the hair cells would result in lowered sensitivity at threshold, but loudness would be comparable to normal at higher intensities.

The indirect tests, which were developed to substitute the direct tests and were partly reviewed earlier,[2] do not necessarily operate on exactly the same anatomical level. Nevertheless, some of their results agree reasonably well with the clinical categories separated on the basis of the direct loudness tests.

Auditory fatigue is one of the phenomena which have been related to recruitment. Indeed, there are certain facts favoring this parallelism: In ears fatigued by intense stimulation a loss appears which shows a characteristic recruitment phenomenon.[3] It is also known that persons with a perceptive type of hearing loss are much more handicapped in noisy surroundings than are normal subjects or patients with conductive hearing loss.

In this paper I have confined myself to studying post-stimulatory auditory fatigue in normal subjects and in cases with various types of hearing loss. The recovery of the threshold after stimulation has been traced only at the stimulation frequency with a self-recording audiometer. The results show that certain ears are much more sensitive to continued stimulation than the normal average. This phenomenon can be used in predicting the subjects likely to develop a permanent hearing loss due to the effect of noise in working places, and, in certain cases, in evaluation of the principal type of hearing loss.

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Review of Earlier Studies

The first serious attempts to determine auditory fatigue were made by Rawdon-Smith,[4-5] and Ewing and Littler,[6] who exposed the normal ear to a two- to four-minute fatiguing stimulus. Results of these two studies seem to suggest that the amount of fatigue is greater at the higher frequencies than at the lower. However, the exact magnitudes are difficult to determine because the measuring points after cessation of the stimulus were far from precise and the individual variations very great.

The use of post-stimulatory fatigue in diagnosis was studied by de Maré[7] with short-tone fatiguing stimuli. Gardner,[8] who also used these types of stimuli, described a precise method for classifying hearing impairments. These two workers were the first to realize the extreme importance of precise intervals between the cessation of the stimulus and the measuring point for threshold recovery. Using longer stimulating tones, with which this paper is concerned, Huizing[9] proposed a simple method for classifying hearing impairments. If the ear was fatigued during three minutes with a 2000 cps 30 db. sensation level (SL) tone, the threshold shift measured "immediately" after cessation of the fatiguing stimulus was from 5 to 12 db. in normal ears. In conductive deafness the corresponding shift was from 7 to 12 db., in perceptive deafness without recruitment from 5 to 11 db., and with recruitment from 19 to 22 db. Huizing concluded that the amount of fatigue in perceptive deafness depends rather on the loudness value of the fatiguing tone than on its intensity.

In all fatigue experiments it is of great value to trace the affected threshold accurately as a function of time. The farther the exact measuring moment is from the end-point of stimulation, the less will be the temporary threshold shift. This automatic recording was employed throughout Hood's[10] investigation, a part of which deals also with post-stimulatory fatigue.

For the lower sensation level exposures, Hood reported data on three subjects stimulated for one minute with a 2000 cps tone at 50 to 110 db. sensation levels. It appeared that fatigue produced 10 seconds after the cessation of the stimulus was nearly equal at 50 to 90 db. levels, of the order of 8 to 10 db. This was followed by a sharp rise in fatigue as the sensation level of the fatiguing tone was further increased.

Lierle and Reger[11] compared the resulting post-stimulatory fatigue by stimulating the ear for one minute at 20 and at 80 db. sensation level. The results showed in three subjects fatigue of about 8 db. for 20 db. sensation level exposures as contrasted with 2.5 to 4 db. fatigue produced with an 80 db. SL tone. This relation was later tested by Hirsh and Burns,[12] and the conclusion reached by Lierle and Reger was modified into the statement that a 20 db. SL stimulation does not produce less post-stimulatory fatigue than an 80 db. SL tone. The fatigue was of the order of 6 to 10 db., measured 15 seconds after cessation of the stimulus, and confirmed the earlier results of Hood.[10] Hirsh and Burns, on the contrary, found no sharp increase in the fatigue after 100 db. SL stimulation; the fatigue was on the even level of the weaker tones.

Hirsh and Bilger[12] explored the fatigue with a 1000 cps tone at 20, 60, 80, 90, and 100 db. SL's for durations varying from 10 seconds to four minutes. The earlier findings of Hirsh and Ward[13] to the effect that the sensitivity is noticeably better after the first minute of the post-stimulatory period and this is followed by a second decrease, a "bounce," were generally confirmed. However, at 20 db. SL stimulation there appeared no bounce effect, but the sensitivity returned steadily to normal.

In an earlier work of Caussé and Chavasse[14] the amount of post-stimulatory fatigue was shown to depend considerably upon the frequency. Their results indicated that with a 30 db. SL stimulation for 30 seconds the fatigue was appreciable only above 500 cps and remained nearly con-

stant (about 4 db.) between 1000 and 8000 cps, measured 25 seconds after the cessation of the fatiguing tone.

Differential Diagnostic Information

De Maré,[7] and Gardner [8] tried to differentiate especially the groups of conductive and perceptive deafness with short-tone fatiguing stimuli. Gardner suggested testing the intensity range up to 80-100 db. level at 20 db. intervals. The shift from the normal curve indicated the amount of conductive deafness, and the remaining part of the hearing loss was due to the perceptive component.

Huizing [9] claimed that with his modification of the test it is possible to pick out the recruiting cases. In these the fatigue should be more than normal, whereas all other groups are comparable to normal.

Hood [10] reported fatigue of 5 to 7 db. on three patients, measured five minutes from the cessation of the 15 to 55 db. SL stimuli. These cases represented end-organ deafness. One case, stimulated with a 10 db. SL tone, showed no fatigue. The tests were made at 2000 and 4000 cps.

Rawdon-Smith [5] maintained that post-stimulatory fatigue is a phenomenon which is partly related to the end-organ and partly due to the intervention of the central mechanism. The central effect observed by Rawdon-Smith [5] was shown by Caussé and Chavasse [14] to be due to "shadow-stimulation" from the opposite ear; they regarded the phenomenon as entirely peripheral in nature.

The different results obtained in recruiting cases were ascribed by Huizing [9] to increased loudness; it would therefore be the loudness and not the intensity level that determines the fatigue pattern at a certain frequency. Hood [10] reasoned that as the fatigue is a phenomenon related to the end-organ, subjects with a diseased inner ear are more sensitive to continued stimulation than others.

There is at present substantial evidence against the assumption that, at least in normal ears, the fatigue is determined by loudness. The results of Hood,[10] and of Hirsh and Bilger [12] suggest that, up to quite high sensation levels (90 db.) the increasing loudness does not introduce any noticeable increase in post-stimulatory fatigue, and Lierle and Reger [11] rather maintain that a low sensation level (and also low loudness) exposure results in greater fatigue than does a higher SL exposure.

Experimental Procedure

For this study I used a 30 db. sensation level fatiguing tone in all cases. There were several reasons for this: a tone of 30 db. SL level is easily tolerated, and there is little fear of causing any deleterious effects even on patients with severe hearing loss. Furthermore, at this level there are earlier data by Huizing [9] in both normal and pathological ears suggesting that the test gives clear differential diagnostic information. The experiments of Caussé and Chavasse [14] also provided data on this particular sensation level as a function of frequency, and demonstrated an increased fatigability in the 1000-8000 cps range as compared with the lower frequencies.

Air and bone conduction audiograms were obtained in the usual manner with a Peters SPD 2 audiometer. The PDR 10 earphones showed reasonably flat frequency response characteristics and were calibrated for a 60 db. setting of the audiometer as well as for a fixed input of 100 mv. for all frequencies. White noise with some preponderane on the middle frequencies was employed as a masking noise according to the principles described earlier.[15]

The test subject was thereafter seated in a testing room for exploration of the fatigue characteristics. The measurements were done with a self-recording Békésy-type audiometer in which the subject regulated the tone intensity in his ear by means of a push button, pressing it as soon as the tone was heard and releasing it as soon as the tone disappeared. The recording was done automatically in the adjacent control room;

TABLE 1.—*Results on Post-Stimulatory Fatigue on Normal Subjects*

		15 Sec.		30 Sec.		60 Sec.		90 Sec.		120 Sec.	
		M, Db.	SD	M, Db.	SD	M, Db.	SD	M, Db.	SD	M, Db.	SD
One test subject, 10 runs	250	7.6		4.2		1.8		2.0		1.3	
	500	9.1	2.3	4.4	1.7	1.0	1.4	0.9	2.0	0.4	1.8
	1000	12.8	2.3	8.7	1.3	4.7	2.2	2.6	1.4	1.9	1.2
	2000	15.1	2.3	12.0	3.3	7.8	2.6	6.4	2.4	6.2	2.6
	4000	15.7	2.0	11.7	2.5	7.4	2.5	5.4	2.1	4.5	2.8
	6000	17.0		12.1		7.8		7.0		6.0	
Group of 12 ears	500	10.9	3.6	6.1	3.5	3.9	2.8	1.0	2.9	1.0	2.8
	1000	13.2	4.2	8.4	4.9	5.6	3.9	4.6	3.3	3.5	2.7
	2000	16.4	4.7	11.9	4.7	7.8	4.1	6.3	3.4	6.5	3.6
	4000	17.3	4.1	13.2	5.0	8.9	3.5	8.9	3.1	7.6	3.3

the rate of attenuation was 140 db/second in 1 db. steps and the speed of the paper was 12 mm/minute.

The subject was first instructed to record his threshold for a one-minute period, and, if the tracing was satisfactory for a stable threshold, a 30 db. sensation level tone was turned on for three minutes. After this period the subject was again instructed to record his threshold with the push button for at least a two-minute period. All recordings were done with continuous tones at the same fatiguing and testing frequency, and no adjacent frequency areas were explored.

In normally hearing subjects the frequencies 4000, 2000, 1000, and 500 were specially tested because of their primary importance in hearing for speech. In some instances values were also recorded at 6000 and 250 cps. The testing was always begun at the highest frequency, proceeding systematically to the lower end in order to avoid any overlapping fatigue of previous frequency testing. In these tests one highly experienced person was used for repeated tests at intervals of a few days to check the consistency of the results. A group of six persons, mainly medical personnel, were used as the main body of subjects to reveal individual variations in the testing results.

In the hard of hearing group only one or two frequencies were explored for the fatigue pattern. At these frequencies, binaural or monaural direct loudness balance tests were made for detailed information of the recruitment function in each of the pathological cases. Technique of these tests has been fully described earlier.[16]

Results

Normal Subjects.—The mean values for post-stimulatory fatigue at various frequencies for the experienced subject and for the 12 ears of other subjects are presented in the Table. The graphic presentation of re-

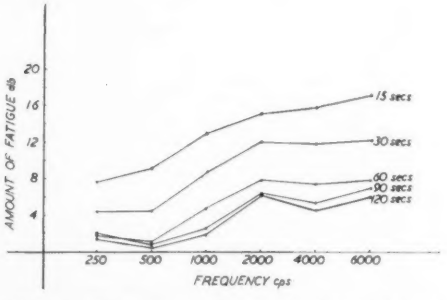

Fig. 1.—The course of post-stimulatory fatigue at frequencies from 250 to 6000 cps. Results show mean values of 10 test sessions on one experienced subject. The time from cessation of stimulus is the parameter. Recovery occurs rapidly during the first minute but is considerably slower in the second half of the testing period.

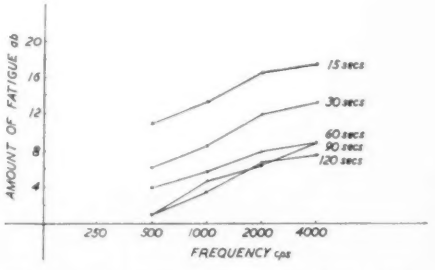

Fig. 2.—The course of post-stimulatory fatigue between 500 and 4000 cps in a group of 12 ears. The time from cessation of the stimulating tone is the parameter. The mean values at 4000 cps are slightly higher than those in Figure 1. Except for the two-minute measuring point, the same is observed also at 500 cps.

sults for both figures, using the time from the cessation of the stimulus as a parameter, appears in Figures 1 and 2.

It is obvious and in accordance with expectation that post-stimulatory fatigue decreases at all frequencies, as the time measured from the cessation of the fatiguing tone increases. However, the rate of recovery does not have an even course but is very rapid shortly after stimulation, and when one minute has been allowed for the recovery the remaining increase towards the normal threshold seems to be quite slow. In this respect there is little difference between the various frequencies.

The individual variability of the post-stimulatory fatigue can be appreciated from the standard deviations (SD) presented in the Table. It is noted that even an experienced subject does not present a unanimous response, but the values for the 10 different test sessions show SD's varying from 1.2 to 3.3. For the 12 ears in the group of observers the SD's are somewhat greater and indicate considerable spread in the amount of post-stimulatory fatigue.

The frequency differences in post-stimulatory recovery become somewhat clearer from Figures 3 and 4, where the fatigue at each frequency is presented as a function of time from the cessation of the stimulus. Figure 3 shows the average results for the experienced subject and Figure 4 for the group of observers. Remembering the standard deviations in the Table, one can see that the recovery curves for 2000, 4000, and 6000 cps in Figure 3 are of the same magnitude and show clearly higher values for post-stimulatory fatigue than the lower frequencies. At 250 and 500 cps, the recovery follows quite rapidly and the post-stimulatory fatigue is nearly negligible after two minutes of recovery. The curve for 1000 cps occupies an intermittent area between these two frequency groups.

In Figure 4 the curve for 4000 cps lies somewhat higher than the 2000 cps curve. However, calculation with the SD's shows that the difference may have arisen by chance. The more the frequency is further lowered, the better is the recovery.

Comparison with Earlier Data for Normal Subjects.—It is a common experience that even in uniform normal material there occurs considerable spread of the results for post-stimulatory fatigue. Thus no single figure can be considered representative of fatigues; rather an area for normal fatigue must be defined. This spread was apparent in many of the earlier results.[4-6,10,12-13,17] The intersubject variability (Table) is also observed in the results of Harris.[18] A certain caution must therefore be exercised before the test is applied in diagnosis, or

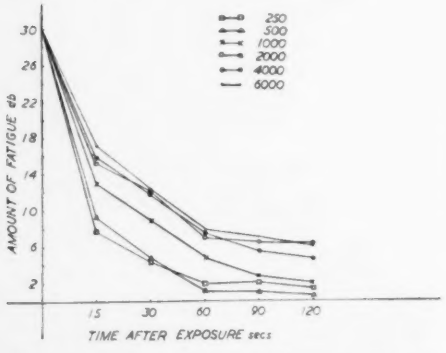

Fig. 3.—Same results as in Figure 1 with frequency as parameter. It is seen that at 2000, 4000, and 6000 cps the recovery function is similar and presents much more fatigue than that occurring at 250 and 500 cps. The curve for 1000 cps assumes an intermediate position.

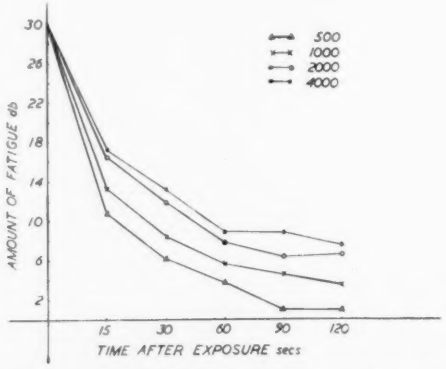

Fig. 4.—Same results as in Figure 2 with frequency as parameter. At the point of two minutes after cessation of the stimulating tone the frequency dependence is quite similar to that in Figure 3.

for disclosing cases especially sensitive to the effect of noise.

There are earlier data on normal subjects,[9-12,14] some of which can be correlated with the present results. The main difficulty lies in the fact that nearly all investigators have used different variables in testing post-stimulatory fatigue. The choice of varying (1) duration of the fatiguing stimulus, (2) its intensity, and (3) its frequency, and (4) the measuring point during the recovery period gives rise to a discouraging number of possibilities for combination. However, using earlier findings[10-12] regarding the development and degree of post-stimulatory fatigue, the three-minute 30 db. SL stimulation employed in the present study can with reasonable accuracy be correlated with studies employing stimulus durations from one to four minutes and sensation levels between 20 and 80 db. Inside these limits the variation in duration and intensity shows only a slight effect on the post-stimulatory fatigue at specific frequencies, and the figures can thus be considered fairly comparable.

In Figure 5 the present results as to post-stimulatory fatigue are compared with some earlier data at 1000 cps. Lierle and Reger's[11] data are computed averages for their three subjects (their Figure 2); Hirsh and Bilger's[12] curve is computed from their Figure 3. The single triangle for Hirsh and Burns is reproduced after their Figure 2; it is curious to note that while this value represents only a one-minute stimulus duration at 20 db. SL, it shows more fatigue than the value after a four-minute stimulation by Hirsh and Bilger. The dot for Caussé and Chavasse cannot be strictly comparable because it was obtained with only 30 seconds' stimulation with a 30 db. SL tone. It appears that, except for the point of 15 seconds after cessation of the stimulus, the data of Lierle and Reger[11] and those of the present study are of the same magnitude, while the curve of Hirsh and Bilger[12] shows clearly smaller values.

A similar graph was reconstructed in Figure 6 for a fatiguing tone of 2000 cps. It is seen that values of other workers must have been expressed mostly at single points as the recovery has generally been recorded only at one moment after stimulation. The triangle of Hirsh and Burns was obtained from their Figure 2. The vertical line expresses the area defined by Huizing[9] as representing the normal range for fatigue. As Huizing did not specify his measuring moment "immediately" after stimulation, it has been assumed to be 10 seconds. The dot of Caussé and Chavasse parallels that in Figure 5 for a 1000 cps tone. The fatigue value obtained by Hood[10] is nearly 8 db. Incidentally, the amount of fatigue by Hirsh and Burns, 9.5 db., was of this same magnitude even after 100 db. stimulation level, while the corresponding figure for

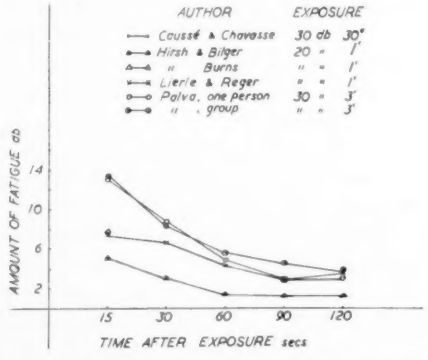

Fig. 5.—The amount of post-stimulatory fatigue at 1000 cps in normal material according to different investigators. For further details see text.

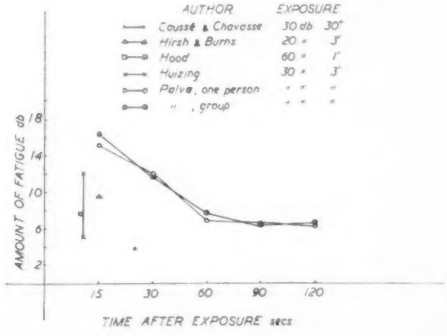

Fig. 6.—The amount of post-stimulatory fatigue at 2000 cps in normal material, according to different investigators. For further details see text.

Hood [10] in his Figure 6, if averaged, would be over 30 db. for the same three-minute stimulus duration.

The phenomenon of bounce found by Hirsh and Ward,[13] and confirmed in a later paper by Hirsh and Bilger,[12] could be observed in a few cases. The sensitivity was some 1 to 3 db. better at 60, or sometimes at 90 seconds, after cessation of the stimulus as compared with the next measuring moment. However, it was observed in less than 10% of all audiograms. It may be that the low frequency for the bounce in the present study is due to the low sensation level.

Harris [17] suspected that the bounce was due to tinnitus in Hirsh and Ward's paper; Hirsh and Bilger therefore employed interrupted tone for tracing the recovery in their later experiments and emphatically stressed that the bounce was real and not due to tinnitus.

In a self-recording audiometer, although the tracing is made with a continuous tone, the procedure is essentially an interrupted tone measurement. This is due to the fact that the subject presses the key to cause the tone to disappear totally; it appears again when the key is released, disappears again, and so on. I am also convinced that tinnitus has nothing whatever to do with the bounce. Slight inattention in listening is a much more likely explanation in some cases, and the presence of tinnitus has an entirely different result (Fig. 7).

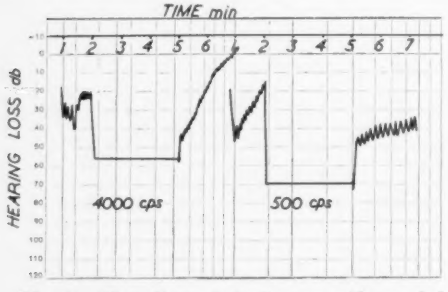

Fig. 7.—The effect of tinnitus on self-recorded curves. In the left curve the subject follows the tinnitus, developed during the stimulation period, until the test is discontinued. In the right, the tinnitus existing before the test is responsible for the steadily rising threshold.

Harris [17] also assumed the presence of tinnitus to be the cause of a slow rate of recovery in Wilson's [19] curves for post-stimulatory fatigue, studied at a higher frequency than the stimulating tone. In his opinion [17] the rate of recovery was much faster, and the threshold rapidly returned to normal if the post-stimulatory testing was made with interrupted tones.

In my experience the effect of tinnitus is entirely opposite. If a subject has tinnitus, it can instantly be seen from the self-recorded graph: the post-stimulatory curve rises ad infinitum as the subject tries to make the tone disappear by pressing the key, but naturally cannot get rid of the tinnitus. During the prestimulatory threshold recording, the effect of tinnitus causes the threshold to rise continuously but not as rapidly as during the poststimulatory period, where the intensity of the tinnitus is generally much greater. These relationships are illustrated in Figure 7.

Hard of Hearing Subjects.—In this study 41 subjects with various types of hearing impairment were used. Pure conductive hearing loss was obvious in 7 cases; in 32 there was recruiting perceptive, and in 2 cases nonrecruiting perceptive deafness. Recruitment testing was done by the direct loudness balance test in all except two cases. In these last, the hearing loss was so flat that even monaural balance tests could not be employed. Conclusions regarding the presence of recruitment were made on the basis of the 2 to 3 db. wide threshold excursions in a continuous tone self-recording audiogram.

In hard-of-hearing subjects the test for post-stimulatory fatigue was always made at the same frequency where the recruitment function was measured. The difference at threshold between the two ears at the testing frequency was in all cases less than 50 db.

On the basis of the recovery curves in Figures 3 and 4 it was decided to use the curve for 2000 cps in Figure 4 as the only reference to which the results in the hard-of-hearing cases were correlated, whether

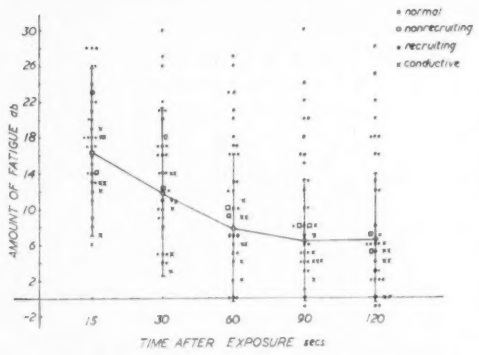

Fig. 8.—Scattergram of individual results of post-stimulatory fatigue at 2000, 3000, or 4000 cps for 37 subjects, belonging to various categories of deafness. The solid line with circles is the normal fatigue curve for 2000 cps from Figure 4. The vertical lines indicate the area of normal deviation of 2 SD's from the mean. The separation of normal and abnormal fatigue values is most marked at the 90- and 120-second measuring points.

the testing frequency was 2000, 3000, or 4000 cps. Recovery curves at 1000 cps and at 500 cps were correlated to the corresponding normal curves in Figure 4. However, the great majority of tests were made at 2000 cps, which therefore presents an exactly similar correlation point to the earlier results of Huizing.[9]

The results in 38 cases tested in the 2000-4000 range are related to the normal 2000 cps curve in Figure 8. In this scattergram the vertical lines crossing the 2000 cps normal curve indicate the area of 2 SD's around the mean value. Values indicating a normal post-stimulatory pattern should rarely deviate from the normal mean more than this amount.

Together with the higher means, the rather large variability causes the first two measuring moments, 15 and 30 seconds after cessation of the stimulus, to be less suitable for picking out susceptible persons than the later measuring moments, especially 120 seconds. If the post-stimulatory fatigue at this point exceeds 14 db., it seems safe to say that one has found an individual differing from the normal more than expected and that this person is liable to more than average suffering in noisy surroundings.

The recovery of conductively deaf persons resembles that of normal subjects. Also in the recruiting perceptively deaf group more than half of the values agree closely with those characteristic of normal recovery. However, they include some showing very poor recovery, and after two minutes of cessation of the stimulus, the threshold may still be about 20-25 db. poorer than at the beginning of the test. Of the two cases without recruitment, both show normal recovery.

The distribution of individual values around the normal 2000 cps curve indicates a fairly even spread at the first three measuring points. At 90 and 120 seconds the normal values may be somewhat high because there are definitely more values below than above the mean, excluding those showing a clearly abnormal result. It may be that the presence of two rather sensitive persons in the normal group of 12 ears has shifted the average slightly higher than it should lie.

When the self-recording audiometer is used, it is obvious that the method of measuring fatigue shortly after cessation of the stimulus[9] does not reveal as many susceptible ears as later measuring moments. For instance, at the point of 15 seconds there are five susceptible ears in Figure 8, but one of them showed normal recovery at later measuring points. This leaves only four ears for the 120 seconds observation point, but at this measuring moment there appear five additional cases showing abnormal fatigue. Huizing's[9] figure, 19-22 db. fatigue as indicator of recruiting deafness, is not at all applicable to the present series, but it must be remembered that the testing techniques were also quite different.

Two cases of recruiting patients were tested at 1000 cps and at 500 cps. The recovery function was comparable to normal in all of them.

Comment

The purpose of post-stimulatory tests has been twofold, i. e., for differential diagnosis

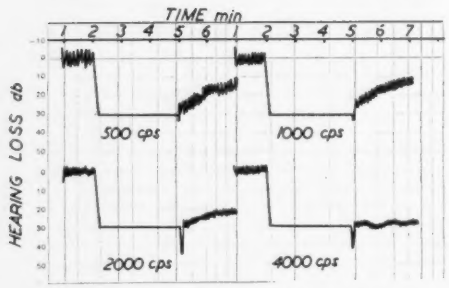

Fig. 9.—Abnormally large fatigue in a normally hearing subject at all frequencies. The amount of fatigue at different frequencies follows closely the relationship observed in Figures 3 and 4.

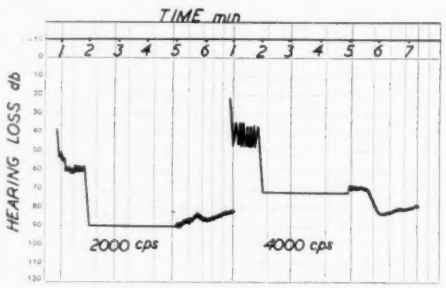

Fig. 10.—Abnormally large fatigue curve in two cases with asymptomatic (left) and delayed (right) recruitment function in perceptive deafness. It is of special interest to note that the initially large excursions, characteristic of delayed recruitment, become very small when the ear is fatigued. The susceptibility of the ear causes the post-stimulatory level to deteriorate even beyond the fatiguing level.

or for picking out those individuals who are more sensitive than the normal average to the effect of continued stimulation.

In normally hearing persons, obviously, the latter is the purpose of the test. The curves in Figures 1 to 4 indicate that the fatigability varies to some extent as a function of frequency, and individually at all frequencies within considerable limits. Therefore one has to allow a rather wide area for the tracings still to be classified as normal. In the preceding chapter it was suggested that post-stimulatory fatigue of more than 14 db. between 2000 and 6000 cps indicates abnormality in the recovery. At lower frequencies the area of normal fatigue is considerably smaller.

Except for those used as a reference, few other normally hearing persons have been tested hitherto. One subject, known to have some subjective difficulties in listening to speech in noise presented quite an interesting picture. The recovery curves in Figure 9 show that there is some recovery at 500 and also at 1000 cps, but at 2000 cps and at 4000 cps the post-stimulatory tracing continues to be nearly 30 db. depressed throughout the recovery recording. It is obvious that this amount of fatigue is far outside normal limits, and if this person were to work in a very noisy place, the ear might suffer considerable damage in a short time.

In the hard of hearing group the question of diagnosis arises first. Remembering the scattergram in Figure 8 one can say that the majority of perceptively deaf persons tested present similar recovery function as normal and conductively deaf cases. The group of abnormal responses includes only recruiting cases, but it is not possible to use the test for finer distinction, as the recovery may also be perfectly normal. The type of recruitment function did not seem to have any appreciable effect upon the recovery: Figure 10 presents a case with asymptomatic recruitment and another with delayed recruitment. They both show abnormally large fatigue figures during the two minutes' recovery period.

It seems easier to discover cases abnormally susceptible to continued stimulation than to make a diagnosis. The measuring points near the cessation of the stimulus seem to be less useful than the points of 90 or 120 seconds after cessation of the stimulus. This is due to the fact that the rather large individual spread of results and higher mean values shortly after stimulation cause an initial overlapping of the abnormal and some of the normal results.

The frequency dependence of post-stimulatory fatigue is obvious. Normally there is only little fatigue at 500 cps and lower after three minutes of 30 db. SL stimulation. At 1000 cps there is considerably more fatigue, and the recovery is slow-

est at 2000 cps and higher frequencies. This relationship is essentially the same as that found by Caussé and Chavasse,[14] although the figures due to different stimulation differ much in magnitude.

Conclusions

The results show that the amount of post-stimulatory fatigue varies with the frequency in that the lower frequencies present less fatigue than the higher. There is also considerable normal spread of results and distinct intersubject variation.

When fatigue values deviating more than 2 SD's from the normal averages are accepted as signs of abnormal fatigability, the rounded upper limit is 6 db. at 500 cps, 9 db. at 1000 cps, and 14 db. at 2000-4000 cps if the fatigue is measured two minutes after cessation of a 30 db. stimulation. However, the values of normal recovery on 26 ears with hearing impairments in Figure 8 suggest that these figures might be somewhat lower. Fatigue of more than 10 db. at 2000-4000 cps might therefore be held as pathological. At 1000 cps the limit could be set at 7-8 db. and at 500 cps at 4-5 db.

In diagnosis the use of post-stimulatory fatigue does not seem helpful beyond separating some perceptively deaf cases from conductive deafness. Many recruiting subjects show normal recovery and some show abnormally great fatigue. In conductive deafness, as well as in normal ears, recovery is as a rule rapid, but in some cases there may be abnormal sensitivity to continuous stimulation.

Testing post-stimulatory fatigue may be useful in indicating a person's ability to tolerate noise in his working place. The cases liable to develop permanent damage to hearing can be predicted and thus kept under close observation.

It seems necessary to make a continuous record of both the prestimulation threshold and the post-stimulatory recovery. It is then possible to follow the recovery function in great detail and to ascertain the reliability of the data.

The effect of tinnitus in an ear to be tested causes the prestimulatory threshold and the recovery curves to rise continuously so that no steady tracing is obtained. This is easily recognizable in the recording; in these quite rare cases the use of interrupted test tones is indicated.

Summary

Tests for post-stimulatory fatigue are made with a self-recording audiometer employing a rate of change of 140 db/minute in 1 db. steps. The prestimulation threshold is traced during one minute stimulation; then a tone of 30 db. SL of the same frequency is introduced for three minutes, and finally the post-stimulatory recovery is traced during a two-minute period.

Normal data for 10 runs on one highly experienced person and for 12 ears are presented between 250 to 6000 cps, while the main studies are limited to the range of 500 to 4000 cps. It is shown that the recovery differs as a function of frequency. Roundly, the upper limits of normal fatigue at two minutes from the cessation of the stimulus are 4-5 db. at 500 cps, 7-8 db. at 1000 cps, and 10 db. between 2000 and 4000 cps.

Forty-one hard of hearing cases were studied by the same test after classifying the cases into different groups by direct recruitment tests and clinical data. Conductively deaf ears show normal recovery. The same was observed in many cases of recruiting deafness and in two subjects with nonrecruiting perceptive deafness, while some of the recruiting cases show abnormally poor recovery. The recovery did not seem to be related to the recruitment function.

It seems that the main value of post-stimulatory tests does not lie in diagnosis but in picking out the cases which are abnormally sensitive to continued stimulation. These persons can be kept under close observation and shifted to less noisy surroundings if permanent hearing loss seems likely to develop.

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REFERENCES

1. Palva, T., and Ojala, L.: Middle Ear Conduction Deafness and Bone Conduction, Acta oto-laryng. 45:137, 1955.

2. Palva, T.: Recruitment Testing, A. M. A. Arch. oto-laryng. 66:93, 1957.

3. Davis, H.; Morgan, C. T.; Hawkins, J. E.; Galambos, R., and Smith, F. W.: Temporary Deafness Following Exposure to Loud Tones and Noise, Acta oto-laryng., Supp. 87, 1950.

4. Rawdon-Smith, A. F.: Auditory Fatigue, Brit. J. Psychol. 25:77, 1934.

5. Rawdon-Smith, A. F.: Experimental Deafness, Brit. J. Psychol. 26:233, 1936.

6. Ewing, A. W. A., and Littler, T. S.: Auditory Fatigue and Adaptation, Brit. J. Psychol. 25:284, 1935.

7. de Maré, G.: Audiometrische Untersuchungen über das Verhalten des normalen und schwerhörigen Ohres bei funktioneller Belastung, nebst Bemerkungen zur Theorie des Gehörs, Acta otolaryng., Supp. 31, 1939.

8. Gardner, M. B.: Short Duration Auditory Fatigue as a Method of Classifying Hearing Impairments, J. Acoust. Soc. America 19:178, 1947.

9. Huizing, H. C.: The Relation Between Auditory Fatigue and Recruitment, Acta oto-laryng., Supp. 78, p. 169, 1949.

10. Hood, J. D.: Studies of Auditory Fatigue and Adaptation, Acta oto-laryng., Supp. 92, pp. 1-57, 1950.

11. Lierle, D. M., and Reger, S. N.: Further Studies of Threshold Shifts as Measured with the Békésy-Type Audiometer, Ann. Otol. Rhin. & Laryng. 63:772, 1954.

12. Hirsh, I. J., and Bilger, R. C.: Auditory-Threshold Recovery After Exposures to Pure Tones, J. Acoust. Soc. America 27:1186, 1955.

13. Hirsh, I. J., and Ward, W. D.: Recovery of the Auditory Threshold After Strong Acoustic Stimulation, J. Acoust. Soc. America 24:131, 1952.

14. Caussé, R., and Chavasse, P.: Études sur la fatigue auditive, Année psychol. 43-44:265, 1942-1943.

15. Palva, T.: Masking in Audiometry, Acta-otolaryng., Supp. 118, p. 156, 1954.

16. Palva, T.: Studies on Per-Stimulatory Adaptation in Various Groups of Deafness, Laryngoscope 65:829, 1955.

17. Harris, J. D.: The Roles of Sensation Level and of Sound Pressure in Producing Reversible Auditory Fatigue, Laryngoscope 64:89, 1954.

18. Harris, J. D.: Recovery Curves and Equinoxious Exposures in Reversible Auditory Fatigue Following Stimulation up to 140 Db. Plus, Laryngoscope 63:660, 1953.

19. Wilson, W. H.: Determination of Susceptibility to Abnormal Auditory Fatigue, Ann. Otol. Rhino. & Laryng. 59:399, 1950.

Tympanoplastic Operations

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Operations for improvement of hearing belong to the special field of modern otosurgery. The credit for the development and accomplishment of the operative technique for improvement of hearing in cases of otosclerosis belongs to Lempert. In cases of chronic otitis media, German otologists (especially Wullstein and Zollner) have developed methods during the last few years which can augment the improvement of hearing besides permitting a removal of the discharge of the ears. These operations are called tympanoplasties.

Tympanoplasties begin with the thorough removal of the pathological changes within the temporal bone. It is necessary to reconstruct a functioning alignment of structures for conduction of airborne sound by using the conditions present in the middle ear and closing or replacing the partially or completely destroyed drum with a free skin graft. Two functioning fenestrae are necessary for postoperative hearing success following tympanoplasty. This means that sound pressure difference and phase difference must exist between the two windows. If this prerequisite is not fulfilled, there cannot be movements of the perilymph and motion of the basilar membrane by the sound pressure; without this, passage of the airborne sound from the middle ear to the inner ear does not occur, and the organ of Corti, a kind of transformer, is not activated; therefore there would be no postoperative improvement of the hearing. The more mobile the two windows in the middle ear, and the greater the pressure differences between them, the greater the hearing improvement.

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Allgemeines Krankenhaus Heidberg (Chief: Prof. Dr. med. Heinz Rollin).

Findings within the middle ear in cases of chronic otitis media may be classified as follows:

Group A: Smaller or larger perforations of the drum following trauma or an old inflammation, but now having a noninflamed tympanic cavity with the ossicular chain intact and absence of recurrent otitis media.

Group B: Chronic otitis media with the ossicular chain intact and freely movable.

Group C: Chronic otitis media with partial damage of the ossicular chain but the presence of a functionally intact ossicular chain with practically undisturbed sound transmission to the inner ear.

Group D: Chronic otitis media with interruption of the ossicular chain but the presence of a mobile stapes.

Group E: Chronic otitis media with absence of the ossicular chain but the presence of a mobile membrane sealing the oval window.

Group F: Chronic otitis media with fixation of the ossicular chain or its remainders by firm adhesions or even secondary ossifications. This means lack of mobility of the oval window.

For best hearing results operative procedures must vary according to the needs of the different groups. But the following techniques must be given attention in all tympanoplastic operations:

1. All of the pathologically changed cells within the temporal bone must be removed.

2. There must be exact classification and identification of the conditions encountered in the middle ear through a strong magnification (a microscope with at least a 6× power).

3. The posterior bony wall of the external auditory canal must be removed as far as possible in order to obtain the best view of the round window niche and to get the best condition for the application of the freely transplanted skin graft.

4. The polypi or granulations from the middle ear must be carefully removed,

especial care being taken to clean the round window niche and the ossicles.

5. The mucosa must be preserved in the middle ear as much as possible. Complete removal results in adhesions and annihilates permanent hearing results. A thin layer of mucous membrane must always be left, sometimes even a thickened mucosa, which may eventually become normal when the pathology of the temporal bone is removed. When no mucosa is left in the tympanic cavity, i. e., when there is complete scar formation or epidermization, then a plastic graft with mucous membrane taken from the oral part of the lower lip must be performed in order to avoid a complete closure of the tympanic cavity. Amnion may be used as well.

6. The Eustachian tube must be in communication with the newly created tympanic space which is separated from the external auditory canal by a freely transplanted skin graft. The undisturbed air passage through the tube must be tested during each tympanoplasty. If there is no air exchange between the tube and the newly created tympanic cavity, or if the exchange is blocked later on, then lack of mobility of the round window membrane following the air resorption will prevent any hearing result.

7. The mobility of the two windows in the middle ear must be tested by careful pressure upon the ossicular chain or its remainders. When there are no ossicles, the test is made by careful pressure on the sealing membrane of the oval window with a blunt instrument. If both windows are movable and mobilization of the perilymph by the sound pressure effect occurs after a tympanoplasty, serviceable hearing can be expected. This mobility can be demonstrated whenever a light reflex appears in the round window niche. This reflex appears in coordination with pressure upon the oval window. It may be further demonstrated by filling the niche of the round window with fluid and then watching the movement of the fluid level as pressure is applied to the oval window. It is a sign of pressure exchange present between the scala vestibuli and the scala tympani. If no movement of the perilymph occurs, which means the window is fixed, we may remobilize the perilymphatic movement by a fenestration of the horizontal semicircular canal later on and then expect a hearing success. This second operation can be performed about three to six months after the first one, if a dry, noninflamed operative cavity and a closed, air-containing, and inflatable tympanic space are present.

8. The freely transplanted skin graft must be on the base without wrinkles and must be cut and prepared carefully, in some instances by cutting out small triangles at the edge, because otherwise it may loosen or wrinkle with new perforations as a result. The epithelium of the drum margin and the adjacent part of the anterior meatal wall have to be carefully removed. They serve as a source of nourishment for the transplanted skin graft. With the disepithelization, the danger of a secondary cholesteatomatous formation is avoided.

Special operative treatment of the individual groups is as follows:

Group A: A tympanoplasty is not necessary, but a drum plastic repair is sufficient. The surroundings of the drum perforation are carefully disepithelized according to the size of the perforation, and a corresponding Thiersch flap taken from behind the ear is applied. Gelatinous tampons, carefully laid against the flap, hold the graft in situ.

Groups B and C (both groups get the same kind of treatment): A modified radical operation must be performed with thorough eradication of the pathological cellular system within the temporal bone, care being taken to preserve a small bridge and to save the ossicular chain. Then the remainder of the drum and its margin must be disepithelized along with the adjacent part of the anterior meatal wall. The next step is the removal of the pathology, polypi, and granulations in the middle ear with careful preservation of the still-present mucous membrane covering the promontory. The round win-

dow niche must be inspected and cleaned. Final steps are catheterization of the Eustachian tube, replacement of the drum, and closure of the tympanic cavity by a freely transplanted skin graft with application on the handle of the malleus over the preserved bridge, the bony facial canal, the disepithelized drum margin, and adjacent anterior meatal wall. The Eustachian tube must be left open and in free communication with the tympanic space.

Group D: A radical operation must be performed with removal of the bridge and the remainders of malleus and incus. Then follows careful work in the tympanic cavity as described above with additional application of the transplanted skin flap upon the head of the stapes.

Group E: A radical operation must be followed by careful work in the tympanic cavity, and the removal of the mucous membrane of a small part of the promontory between the oval and round window. This part is used as an additional source of nourishment of the freely transplanted skin graft which has to be applied here too. We must spare the oval window out of the plastic flap in order to avoid minimizing the mobility of the sealing membrane of the oval window.

Group F: A tympanoplastic operation must be performed according to the needs of this type of group. The horizontal semicircular canal is burnished but not opened up, as preparation for a fenestration operation to be performed later on. This may be done after three to six months, provided the operative cavity is epidermized and dry, and the newly created tympanic space is air-containing and closed from the external ear. Then only the part of the flap covering the horizontal canal is lifted; the new fenestra is made over the ampulla of the semicircular canal and covered again by the flap laid back as known from revisions in cases of bony closure of a fenestrated ear.

The training and experience of the surgeon must determine whether he chooses an endaural or a retroauricular approach. Both ways are possible. Either a thick Thiersch flap or a retroauricularly excised and thinned skin graft is used as a freely transplanted graft. The latter has the advantage that it does not roll in at the edges or shrink later on because of the lack of elastic fibers. A thin skin flap excised and taken from the inner side of the upper arm can be used as well. After the operation, antibiotic protection is administered for one week. The first dressing, apart from changing the external dressings, take place on the seventh day after the operation. Only the operative cavity is inspected. Secretion, if present, is sucked away, and a strip of gauze with an antibiotic solution is introduced. From the 9th to the 11th postoperative day, the plastic flap may be disepithelized, i. e., the removal of the loosened upper layer of the epithelium can be performed. Utmost care has to be exercised not to perforate or lift the underlying plastic flap, which usually has a fresh rosy appearance. Careful inflations of the tube start from the 10th postoperative day. These inflations have to be repeated regularly. When one watches with a microscope, movement or bulging out of the transplanted skin graft indicates the communication of the tympanic space with the Eustachian tube. Listening with an auscultation tube is not sufficient. If the Eustachian tube is not successfully inflated, because of swollen mucous membrane, etc., fractioned x-ray treatment of a total of 7-10×100 r, just as an inflammation dose (100 r two times a week) may restore the communication between the tube and the tympanic space. In cases with inclination toward the formation of granulation tissue in the operative cavity, this treatment is serviceable too.

The further treatment of the operative cavity does not differ from the well-known treatment following a radical operation. If a hearing success that is gained by a tympanoplasty regresses to an unserviceable hearing level during the course of aftertreatment, i. e., if a fixation of the ossicular chain, its remainders, or of the oval window mem-

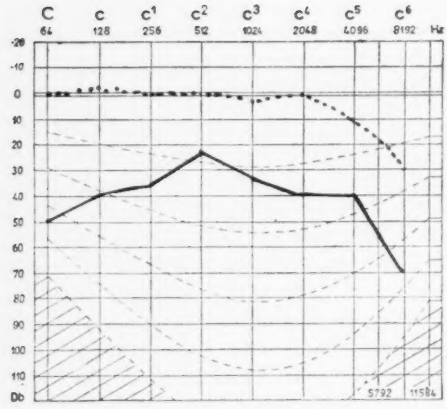

Fig. 1 (Case 1).—Preoperative audiogram.

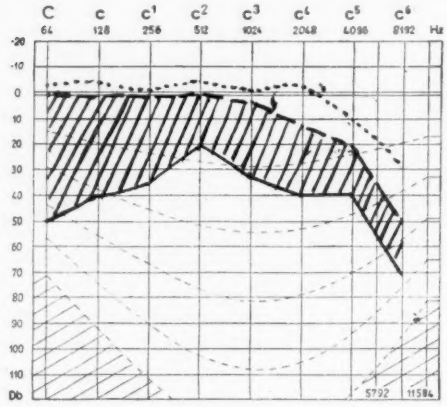

Fig. 2 (Case 1).—Postoperative audiogram.

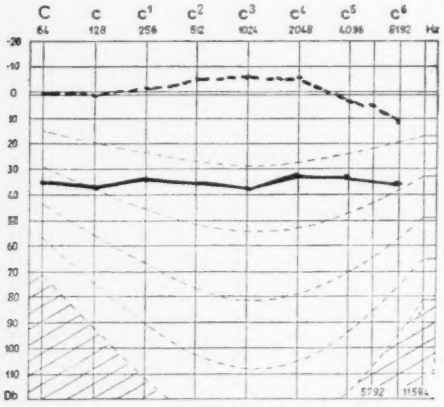

Fig. 3 (Case 2).—Preoperative audiogram.

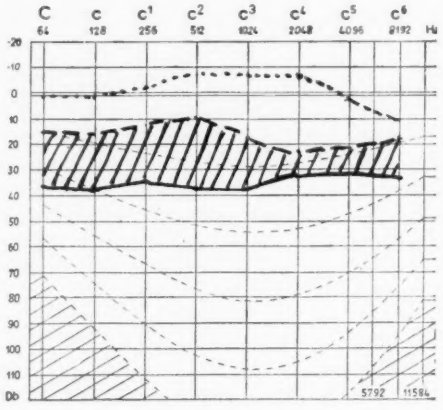

Fig. 4 (Case 2).—Postoperative audiogram.

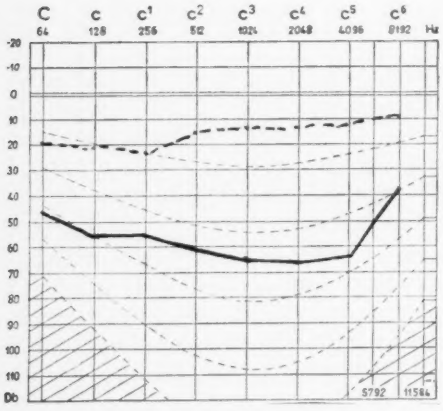

Fig. 5 (Case 3).—Preoperative audiogram, right ear.

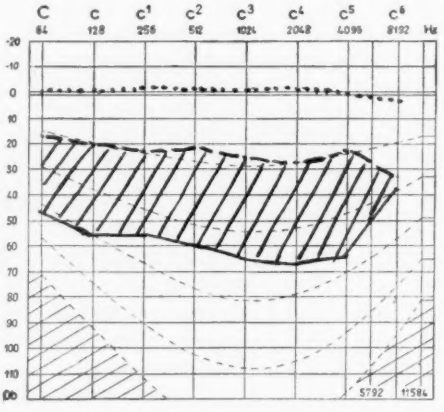

Fig. 6 (Case 3).—Postoperative audiogram, right ear.

brane (secondary case group F) takes place by formations of adhesions etc., a fenestration operation later on may finally raise the hearing to the practical level.

Tympanoplastic operations are difficult procedures, and the utmost concentration, technique, and experience are required from the surgeon. Only otologists who have many opportunities of doing temporal bone surgery should perform this kind of operative treatment. Correctly done, tympanoplastic operations represent an essential enrichment of otosurgery and can often restore the lost hearing in cases of chronic otitis media.

CASE 1.—Patient: woman, 27 years old.

Hearing loss because of a flap against the left ear.

Findings: Central perforation of the left drum; normal mucosa within the middle ear; no discharge.

Hearing: 2 m. for whispered voice; 3.5 m. for loud voice.

Therapy: A drumplasty was performed Feb. 1, 1956. The postoperative course was uneventful. Normal hearing since the operation.

Postoperative hearing: more than 5 m. for whispered voice.

CASE 2.—Patient: youth, 16 years old.

Recurrent discharge of the right ear since his early childhood.

Findings: Central perforation of the drum with mucoid discharge.

Hearing: 1.5 m. for whispered voice; 5 m. for loud voice.

Therapy: A tympanoplasty was performed Nov. 5, 1956. The ossicular chain was intact and freely movable. A small bridge was left. Good pressure exchange between scala vestibuli and scala tympani. Closure of the tympanic space with a freely transplanted skin graft. Uneventful healing of the operative cavity.

Postoperative hearing: more than 5 m. for whispered voice.

CASE 3.—Patient: girl, 13 years old.

Has had discharge of both ears since birth with poor hearing; could not follow the lessons in school because of her hearing loss.

Findings: 0 for whispered voice; 1.5 m. for loud voice (the hearing was equal in both ears).

Therapy: A tympanoplasty of the right ear was performed July 16, 1956, of the left ear Aug. 8, 1956.

Findings: Right ear: Remainders of the malleus and incus were present; the crura of the stapes were destroyed; the footplate of the stapes in situ and movable. Good pressure exchange between the oval and round window. Closure of the tympanic space with a freely transplanted skin graft.

Left ear: Malleus and incus were destroyed; stapes in situ; positive pressure exchange between the oval and the round window. Closure of the tympanic space with a freely transplanted skin graft. Uneventful course of healing. The ears became completely dry. The patient has no more difficulty in school, can follow the lessons. (Note the improvement of the postoperative bone conduction.)

Postoperative hearing: 5 m. for whispered voice, right ear; 4 m. for whispered voice, left ear.

CASE 4.—Patient: woman, 33 years old.

Otitis media at the age of 12 years. Has had recurrent otitis media since then.

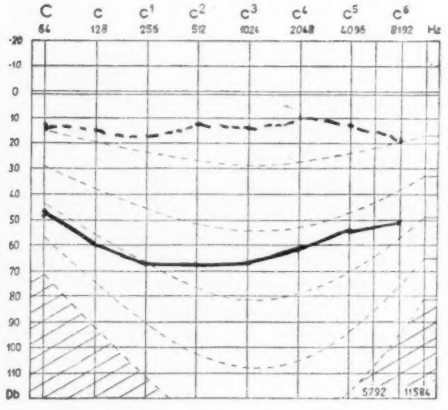

Fig. 7 (Case 3).—Preoperative audiogram, left ear.

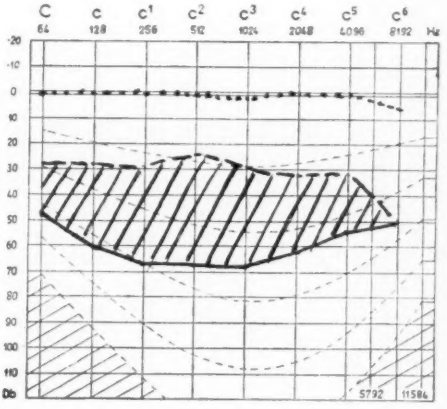

Fig. 8 (Case 3).—Postoperative audiogram, left ear.

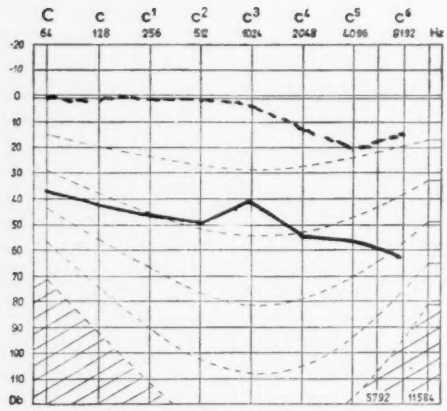

Fig. 9 (Case 4).—Preoperative audiogram.

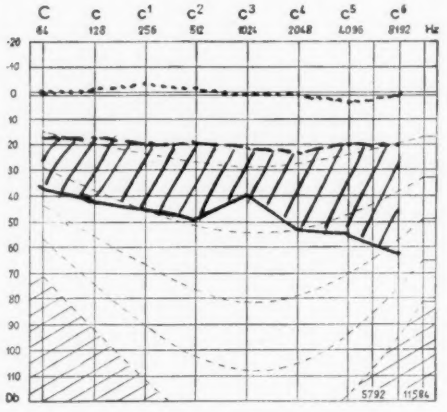

Fig. 10 (Case 4).—Postoperative audiogram. Note the improvement in the postoperative bone conduction line.

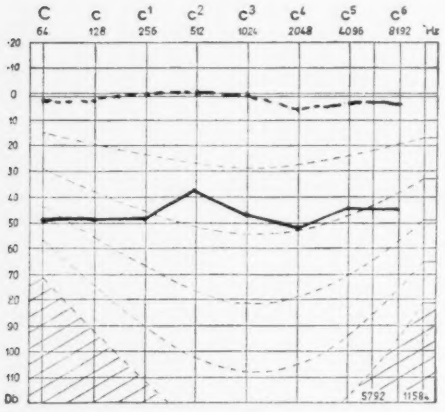

Fig. 11 (Case 5).—Preoperative audiogram.

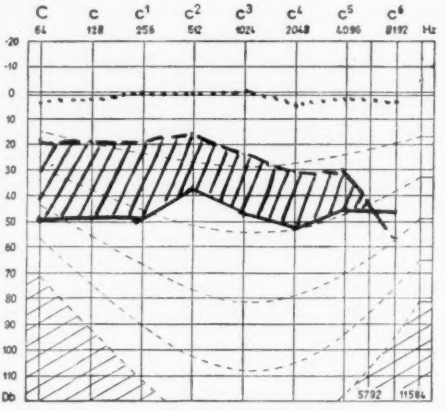

Fig. 12 (Case 5).—Postoperative audiogram.

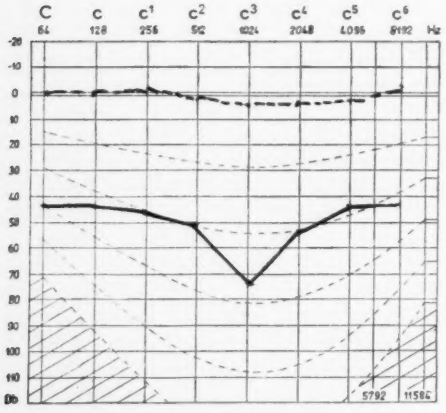

Fig. 13 (Case 6).—Preoperative audiogram.

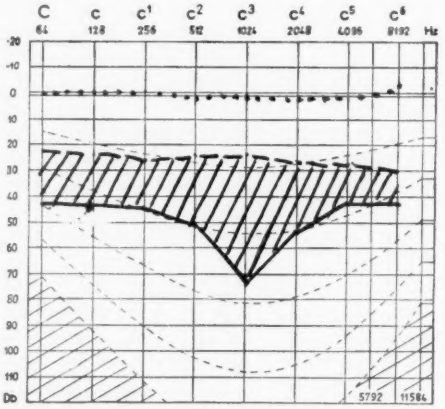

Fig. 14 (Case 6).—Postoperative audiogram.

Findings: Central perforation of the right drum. No discharge at present.

Hearing: 1.0 m. for whispered voice; 2.5 m. for loud voice.

Therapy: A tympanoplasty was performed Sept. 12, 1956. The long process of the incus was destroyed; the stapes was intact and mobile. The malleus and the rest of the incus were removed. Closure of the tympanic space with a freely transplanted skin graft. Postoperative course uneventful.

Postoperative hearing: 5 m. for whispered voice.

CASE 5.—Patient: boy, 14 years old.

Discharge of the left ear since early childhood.

Findings: Marginal perforation of the posterior-superior part of the drum; cholesteatoma present.

Hearing: 0.3 m. for whispered voice; 0.5 m. for loud voice.

Therapy: A tympanoplasty was performed May 7, 1956. There were only remainders of the malleus and incus; the stapes was completely destroyed. Removal of the remainders of the ossicles. Good pressure exchange between the oval and round window. Closure of the tympanic space with a freely transplanted skin graft. Uneventful postoperative course.

Postoperative hearing: 3 m. for whispered voice; more than 5 m. for loud voice.

CASE 6.—Patient: woman, 44 years old.

Patient had measles as a child with drainage of the ears and hearing loss. The discharge of the ears had stopped; the hearing loss has remained.

Findings: Central perforation of the left drum with adhesions to the promontory; scarification of the rest of the drum.

Hearing: A. C. for whispered voice; 0.5 m. for loud voice.

Therapy: A tympanoplasty was performed July 23, 1956. The ossicles were completely fixed by formation of new bone and adhesions between the long process of the incus and stapes to the bony facial canal and the labyrinth. Adhesions in the hypotympanum and the niche of the round window were removed. The adhesions and bone formation between the ossicles and the bony facial canal were not touched. The labyrinth (horizontal semicircular canal) was prepared for the secondary fenestration later on. Normal healing of the operative cavity, which became completely dry and epithelized. Fenestration operation Feb. 4, 1957.

Postoperative hearing: more than 5 m. for whispered voice.

All the patients have maintained their hearing since their discharge from the hospital.

Allgemeines Krankenhaus Heidberg, Hamburg-Langehorn 2.

Abstracts from Current Literature

Ear

CARBONIC ANHYDRASE AND INTERNAL EAR. JORGE CORVERA, Ann. Otol. Rhin. & Laryng. 65:351 (June) 1956.

This paper, based on six patients, constitutes a preliminary report on the treatment of Ménière's disease with acetazolamide (Diamox). The drug was administered in doses of 250 mg. daily, for a period of 21 to 63 days. The ages of patients varied from 24 to 47 years. Complete neurological examinations, as well as vestibular caloric studies, were performed. In all cases, the vertigo disappeared in from three to eight days. Four of the six patients showed improvement in hearing.

The author hopes this small series will stimulate interest in the use of acetazolamide in the treatment of labyrinthine hypertension.

STEELE, Kansas City, Kan.

ATTICOMASTOIDECTOMY. F. W. DAVISON, Laryngoscope 67:191 (March) 1957.

The operation is used for intractable chronically discharging ears with marginal or attic perforations. Cholesteatoma or other complications are of course operative indications also.

Healing time averaged eight weeks. Two out of 122 required a secondary radical mastoidectomy. Skin grafts were not used. The instillation of boric acid powder regularly during the operative period hastened healing.

Flaps are cut and fitted carefully. The aim is to produce what Wullstin calls the Type III Tympanoplasty. The flap in this is placed in contact with the head of the stapes and extends below to form a middle ear cavity.

The Eustachian tube should be open and adhesions carefully removed from the round and oval windows. With these objectives accomplished the resultant hearing should be improved. A table is included showing the pre- and posthearing loss and whether or not the incus was present. Some case reports are shown to illustrate the value of the operation.

HITSCHLER, Philadelphia

DISCUSSION ON NOISE AND ITS EFFECTS. J. D. HOOD, Proc. Roy Soc. Med. 50:217 (April) 1957.

Noise is unwanted sound, and may cause structural damage to the cochlea. It can be transient, periodic, or aperiodic. Transient sounds consist of short duration changes of pressure; periodic are repetitive over a fundamental period; aperiodic show no particular period of recurrence. Sound intensity can be measured in the laboratory by special instruments, setting up a standard of 1000 cps as a basis of measurements. This is likely to be found in a quiet office, but in a busy street it may be 1,000,000. Thunder may amount to almost a billion! Sensitivities of the human ear can be measured. They will, of course, vary for each individual. The article shows drawings of instruments and tables of results. Six subjects were investigated for purposes of this report. In all six, with only slight variations, the curves followed the same general course. The effect of noise upon voice level begins at about 20 db. Then the voice level increases to some 90 db. After that the voice seems unable to match the noise produced. Considerable effort on the part of the subject was required to maintain the voice level after the noise stimulus had been removed. Some conclusions may be drawn from this in re paracusis Willisii. This is a new field for research in which physics, physiology, and psychology play a prominent part.

Continuing the symposium, Prof. W. Burns of the Department of Physiology of the Charing Cross Hospital Medical School spoke of Noise and Its Effects: Physiological Aspects. He said that aural damage from sound is dependent on the kind of noise, its intensity and duration. It is also related to the kind and degree of cochlear function. Physiological criteria are based on electrical potentials known as cochlear microphonic (CM) and the action potential of the auditory nerve (AP). These items have to do with movements of the basilar membrane. The highest frequencies reach their maximum amplitude at the base of the cochlea. Frequent reference is made to the work of Americans, Wever and Bray, who worked in the laboratories at Princeton University some years past. The present need is for more and better understanding of the physiologic process underlying the reaction of the human ear to acoustic

stimuli. This is a deeply involved subject.

At this juncture Mr. D. E. Broadbent of the Applied Psychology Research Unit at Cambridge held forth on Noise and Behavior. The effect, for example, of impaired hearing on the kind of voice one discloses. "The average man of 50 has . . . lost 20 db. at 4000 c/s according to several large-scale surveys." Deafness due to a particular frequency band is rather below that at which the deafness occurs. Distortion of the loudness of sounds is due to the cerebral interpretation of the stimuli sent from the cochlear (auditory) nerve.

ABSTRACTOR'S COMMENT: These researches belong rather in the field of "pure science" than in clinical otology. The patient is ill adjusted to laboratory studies—he has deafness and noises in the head which sometimes lead him to question whether he is subject to his own bodily disturbances or to those brought to him from the external world. He wants to know what you can do about it. Well, what can you?

I. W. VOORHEES, New York.

PERCEPTIVE DEAFNESS WITH GRADUAL LOW TONE LOSS. KENJI NAKAMURA, Japanese J. Otolaryng. 58:1269 (Nov.) 1955.

Perceptive deafness is characterized by impairment of high tones. Numerous reports have already been made on this phenomenon. However, the instances of perceptive deafness with low tones may be found in cases of Ménière's disease or endolymphatic hydrops. Nakamura records four cases of perceptive deafness with low-tone loss. Two had continuous tinnitus. Vestibular test was normal in all cases.

The cause of this type of deafness has been explored in relation to Ménière's disease. Williams regarded it as a kind of "endolymphatic hydrops without vertigo." The existing theories of Yamakawa, Rollin, Lindsay, Fowler, Hilger, Mygind, Lempert, and Hallpike, however, are not able to explain satisfactorily the development of hearing impairment in these cases.

An experimental study was made to discover the cause of such perceptive deafness with gradual low-tone loss.

The author observed the development of gradual low-tone loss in air and bone conduction when auditory fatigue was caused by such noise as filtrated by a pass filter of 200–400 cps. Nakamura inferred the possibility that this type of deafness has its cause in the peripheral neural elements of the auditory organ.

HARA, Los Angeles

INCIDENCE OF CLINICAL OTOSCLEROSIS IN JAPAN. S. HORIGUCHI; Y. OUCHI, and G. TSUNODA, Jap. J. Otolaryng. 58:1289, 1955.

The authors recorded the incidence of clinical otosclerosis which were observed at the Tokyo Medico-Dental Institute during the past three years.

It constituted 1.12% of all cases of impaired hearing. There were 11 male and 12 female cases in this series. The characteristics of otosclerosis were progressive bilateral deafness and tinnitus. The audiograms are similar to those reported by American and European otologists. Diagnosis is made by the blocked bone conduction test and installed air conduction.

HARA, Los Angeles

STUDIES ON DENSITY, CENTER OF GRAVITY AND AXIS OF ROTATION OF HUMAN AUDITORY OSSICLES. Y. SHINOHARA, and Y. SATO, Japanese J. Otolaryng. 59:89 1956.

The density and center of gravity of the auditory ossicles play an important part in the problem of sound conduction.

1. The authors used normal human cadaver specimens of auditory ossicles between the ages of 10 days and 60 years.

2. Density of the incus and malleus was compared with that of water. Density of the combined ossicles was 2-2.8 times that of water. But the density of the malleus was different at its head, neck, and handle. The neck of the malleus weighed heaviest, being 4.0.

3. Each ossicle was suspended at three different points, using the strand of spider web. The suspended bone was rotated once in every three seconds and photographed in order to determine the center of gravity and axis of rotation.

4. Upon vibration the straight line which runs through the centers of gravity of the malleus and the incus does not correspond to the axis of rotation of the system.

HARA, Los Angeles

Larynx

ON CIGARETTE SMOKING, BRONCHIAL CARCINOMA AND CILIARY ACTION: IV. CILIARY STREAMING THRU THE LARYNX AND DISTRIBUTION OF LARYNGEAL EPITHELIUM. A. C. HILDING, Ann. Otol. Rhin. & Laryng. 65:736 (Sept.) 1956.

Studies on ciliary streaming within the tracheobronchial tree and larynx, utilizing human and animal specimens removed after death, reveal some interesting features.

The ciliary streams from the major bronchi are divided into posterior and anterior streams by the carina. Both streams are then directed upward to the subglottic region of the larynx. The posterior stream passes directly between the arytenoids to the rim of the larynx, while the anterior stream passes to the anterior commissure where the squamous epithelium acts as a barrier. The anterior stream divides in two parts, each passing backward along the undersurface of the vocal cord to the interarytenoid space.

Hilding suggests the possibility that laryngeal carcinoma may occur more commonly on the anterior portion of the vocal cords because of tracheobronchial mucus containing carcinogenic substances slowing and dividing in this region of the larynx.

Another relation of ciliary streaming to the distribution of carcinoma might lie in the field of transplant. It seems possible that cells loosened from laryngeal cancer might find their way into the tracheobronchial mucus. As it has been noted by some observers that recurrences following laryngectomy are apt to be found just below the tracheostomy, it is possible that this opening might act as an obstruction in the ciliary stream along the anterior tracheal wall. Detached cancer cells might be carried to this point and transplanted.

The author denies that final conclusions can be drawn from present evidence on these two interesting possibilities.

STEELE, Kansas City, Kan.

CONTACT ULCER OF THE LARYNX. CHARLES C. WOLCOTT, Ann. Otol. Rhin. & Laryng. 65:816 (Sept.) 1956.

Wolcott points out the role of stress in the production of contact ulcer granuloma of the larynx in the patient who has faulty voice production. It is believed that stress causes excessive neural stimulation of the parasympathetic nervous system which excites the vagus. This results in increased strength and excursion of cord action, causing traumatic "hammer and anvil" effect.

The use of anticholinergic drugs, such as methanthelene bromide, which have a ganglionic blocking action have been of value in the treatment of this disorder in the author's small series.

STEELE, Kansas City, Kan.

PARALYSIS OF THE VOCAL CORD: REPORT OF A CASE OF UNILATERAL PARALYSIS WITH INADEQUATE AIRWAY. WILLIAM C. THORNELL, Ann. Otol. Rhin. & Laryng. 65:945 (Dec.) 1956.

Thornell reports a case of unilateral vocal cord paralysis treated successfully by transoral intralaryngeal arytenoidectomy, apparently the only such instance on record. The paralysis was a complication of thyroid surgery and involved the right vocal cord. Following arytenoidectomy, a satisfactory 5 mm. opening was produced between the vocal cords at the widest angle, and the voice, although slightly weak, was adequate.

STEELE, Kansas City, Kan.

CARTOID BODY TUMOR OF THE TRACHEA. MICHAEL S. ZEMAN, Ann. Otol. Rhin. & Laryng. 65: 960 (Dec.) 1956.

This is a report of a case of carotid body tumor occurring in a man 45 years of age. The neoplasm was found in the left upper portion of the trachea, apparently the first such report in the medical literature. It is felt that it originated from carotid body tissue misplaced in the tracheal wall. Hemoptysis was the patient's presenting complaint. The tumor was removed by cup forceps and the base electrocoagulated. After three years, there has been no recurrence.

STEELE, Kansas City, Kan.

CERVICAL METASTASES FROM SQUAMOUS CELL CARCINOMA OF THE LARYNX. ARTHUR J. KUHN; KENNETH D. DEVINE, and JOHN R. MCDONALD, Laryngoscope 67:169 (March) 1957.

Most metastases of carcinoma of the larynx arise by venous spread to the cervical region. This study is based on 602 cases of squamous-cell carcinoma seen at the Mayo Clinic in a five-

year period, 1945-1949, and an attempt is made to determine what influence various factors had on the incidence of cervical metastasis.

The tumors were classified as to site of origin. Nine areas were considered.

Factors carefully analyzed are age, sex, histologic grade, site, correlations (between factors), and duration of symptoms. These various types of tumors are analyzed; criticism of the methods used and comparison of the results with those of others are discussed. There were no significant differences found between the general findings in this work and those in other reports.

Laryngeal cancer in certain locations so frequently metastasizes that cervical lymph nodes should be removed at the time of the original operation, even if the nodes are not palpable. Also, the histologic grade and site of the tumor are most important regarding cervical metastasis, while age, sex, and duration of symptoms are not.

HITSCHLER, Philadelphia

Nose

THE VIRUS THEORY OF NASAL POLYP ETIOLOGY AND ITS PRACTICAL APPLICATIONS. FRANCES L. WEILLE, and ROBERT S. GOHD, Ann. Otol. Rhin. & Laryng. 65:443 (June) 1956.

The studies conducted by the authors show that polyps removed from humans, when put in suspension, are capable of inciting chemical changes in the allantoic fluid of the chick embryo. Increased protein content of that fluid is thereby produced. This phenomenon is capable of being serially transmitted for at least five chick embryo passages.

However, one disturbing feature of the transmission experiments is the loss of virulence of the inciting agent with successive passages through the embryos. It was demonstrated that the incitant, following filtration, retains almost all of its activity. Attempts to produce gross or microscopic tissue changes in the nasal mucosa of experimental animals or in the chick embryos have not been successful.

It is concluded that the experiments suggest a possible relationship between virus and nasal polyposis.

STEELE, Kansas City, Kan.

MANAGEMENT OF NON-MALIGNANT GROWTHS IN THE MAXILLARY SINUS. O. E. VANALYEA, Ann. Otol. Rhin. & Laryng. 65:714 (Sept.) 1956.

Antral cysts and polyps frequently occur and many times go unrecognized. They are best shown by instillation of radiopaque material into the antrum followed by the making of roentgenograms.

Cysts are usually indicated on the roentgenogram by smooth, round shadows arising from the floor of the sinus, while irregular filling defects are more often caused by antral polyps. Inflammatory or allergic sinusitis usually produces a smooth thickening of the antral mucosa.

Frequently it is possible to eliminate cysts by puncture during inferior meatal antral lavage or through an intranasal antrostomy opening. Polyps require the Caldwell-Luc surgical approach for their removal. Choanal polyps commonly arise in the maxillary sinus.

STEELE, Kansas City, Kan.

EXPERIMENTAL TREATMENT OF RECURRENT CARCINOMA OF THE NASOPHARYNX WITH ELECTRODESICCATION, RADIOACTIVE COBALT AND X-RAY IRRADIATION. FRANCES SOOY, Ann. Otol. Rhin. & Laryng. 65:723 (Sept.) 1956.

Sooy reports a method of treating recurrent nasopharyngeal carcinoma by intranasal septectomy, electrodesiccation of the tumor area, and insertion of radioactive cobalt.

The entire posterior three-fourths of the nasal septum is removed, the edges of the defect being electrocoagulated for hemostasis. The tumor area in the nasopharynx is then thoroughly electrodesiccated, and a Foley catheter containing radioactive cobalt is inserted and held in place by anterior and posterior nasal packing.

Of six patients so treated, three are well and free of recurrence for as long as eight years; two died within 13 months of surgery, and one died in 8 months from extension of cervical metastases before operation.

STEELE, Kansas City, Kan.

ANESTHESIA OF THE CHEEK: AN EARLY SIGN OF CARCINOMA OF THE MAXILLARY SINUS. J. G. WALTNER, and RICHARD H. FITTON, Ann. Otol. Rhin. & Laryng. 65:955 (Dec.) 1956.

Hope for improving the five-year cure rate for carcinoma of the antrum must be realized in earlier diagnosis and in the development of newer treatment techniques. The disease is

rarely diagnosed before there is roentgen evidence of bony destruction by the neoplasm. The presence of anesthesia of the maxillary division of the trigeminal nerve should suggest antral malignancy when there is persistent pain, numbness or swelling of the upper jaw, even though the roentgenogram fails to show bony destruction. In such instances, exploration of the antrum is justified and advisable.

STEELE, Kansas City, Kan.

Miscellaneous

SECRETORY SIALOGRAPHY IN DISEASES OF THE MAJOR SALIVARY GLANDS. I. M. BLATT; PHILIP RUBIN; A. J. FRENCH; JAMES H. MAXWELL, and JOHN F. HOLT, Ann. Otol Rhin. & Laryng. 65:295 (June) 1956.

The authors present a number of well-documented cases of inflammatory or neoplastic disease of the major salivary glands. The text is well illustrated by excellent sialograms. Of 135 cases in the series, 40 were normal; there were 56 showing inflammatory disease, and 39 with neoplasm.

Calculi and stricture were the principal causes of chronic obstructive sialodochiectasis, which involves the main duct of the salivary gland. On the other hand, the abnormalities were noted mainly in the peripheral ducts in chronic nonobstructive sialodochiectasis. This group includes Mikulicz's disease, Sjögren's syndrome, and recurrent pyogenic parotitis in adults and children.

By study of the filling and emptying phases of secretory sialography, sialectasis is classified into four stages: punctate (early stage); globular (moderately advanced stage); cavitary (advanced stage); destructive (end stage). Secretory sialography well demonstrates tumors extrinsic to the salivary glands by showing prompt emptying following stimulation of the glands. Intrinsic salivary neoplasms, however, show retention of contrast material on post-evacuation films.

Although sialography is not a new technique, the use of secretory sialography, as developed by the authors, has enhanced the value of this diagnostic and interpretive aid.

STEELE, Kansas City, Kan.

THE CORTICOSTEROIDS IN OTOLARYNGOLOGY. AUBREY G. RAWLINS, Ann. Otol. Rhin. & Laryng. 65:318(June) 1956.

Rawlins cites a number of otolaryngic conditions where administration of corticosteroids is of value: hay fever, status asthmaticus, drug reactions, angioneurotic edema, acute laryngeal edema, critically ill and toxic patients, histamine cephalgia, contact ulcer of the larynx, laryngeal and nasal polyps, early stricture of the esophagus, eczema and dermatitis of the external auditory canal, Bell's palsy, Ménière's disease, preoperative and postoperative patients with depressed adrenal cortices, allergic rhinitis, postsurgical edema, status lymphaticus, anaphylactic and other forms of shock.

It is pointed out that the corticosteroids might be expected to aid the patient with a poor stress mechanism. If intermittent low dosage of corticosteroid therapy were employed, there would be no depression of the adrenal cortex according to the author. He suggests a dosage of 1 to 2 mg. of hydrocortisone twice daily.

STEELE, Kansas City, Kan.

UNUSUAL BRONCHIAL TUMORS. F. E. DONOGHUE; HOWARD A. ANDERSEN, and JOHN R. MCDONALD, Ann. Otol. Rhin. & Laryng. 65:820 (Sept.) 1956.

Fourteen unusual bronchial tumors consisting of five hamartomas, four lipomas, four fibromas and one fibrosarcoma are reported.

The neoplasm was bronchoscopically visible in 10 cases and was removed through the bronchoscope in 4 instances. Lobectomy was performed for removal in six patients, while pneumonectomy was necessary in two cases. Two instances of extrabronchial extension of lipoma were found.

STEELE, Kansas City, Kan.

THE EFFECT OF THYROIDECTOMY ON THE NASAL MUCOSA OF EXPERIMENTAL ANIMALS. G. O. PROUD, and R. D. LANGE, Laryngoscope 67:201-207 (March) 1957.

It is well known that hypothyroidism causes symptoms referable to the ear, nose, and throat. The nasal mucosa seems particularly to be affected. Sometimes the deficiency is too minimal to be measurable by the standards test, but administration of thyroid produces improvement in the patient.

Nasal mucosa of dogs was studied before and after both partial and complete thyroidectomy. With partial thyroidectomy the mucosa soon became thick, and there was a loss of the cilia. With complete thyroidectomy there was in addition metaplasia, inflammatory infiltration, and swelling of the collagen fibers.

The changes occurred very soon after the thyroid deficiency was produced. The loss of cilia would cause less healthy and less physiologic mucous membrane; particularly perhaps the loss of the cilia would predispose to secondary infection.

HITSCHLER, Philadelphia

EXPERIMENTAL STUDIES ON NASAL CILIA. HIROSHI SHIMIZU, Clin. Otolaryng., Kyoto. 48:35, 1955.

In a detailed study of ciliary action the author used nasal mucosa of man, rabbit, and frog and examined the ciliary motion with stroboscopic apparatus and the phase microscope.

The effective stroke, the forward movement of the cilia, is much faster and more vigorous than the recovery stroke. This type of motion described by an individual cilium is repeated by the neighboring cilia with a phase shift, so that the characteristic regular propagation of the wave occurs, presenting a picture resembling a wheat field blown by the wind. This well-established fact is clearly demonstrated by the author's device of observation. Detailed measurement was also possible.

The frequency of the ciliary stroke in man is from 8 to 12 per second. The cleansing in the nose can be ascribed to the movement of the mucous blanket rather than to ciliary motion itself. The author deposited minute particles of cosmetic powder or carmine powder upon the surface of the nasal mucosa and measured the speed and pathway of the mucous transport. The speed of the mucous blanket in the anterior third is slower. This is accounted for by the fact that the mucosa is not ciliated. In the posterior two-thirds of the nose the mucous blanket moves rapidly and may reach 10 mm. per minute. In the human nose the direction of the mucous flow is backward, and the streaming is not always in the straight line to the pharynx.

The determination of temperature effects was made on freshly removed human mucosa and checked against similar ones on living rabbits. The most powerful damage to the ciliary function is caused by drying. The drying needs only to continue for about six minutes to destroy the ciliary activity. A fresh supply of mucous membrane does not recover the cilia once when it is damaged.

The speed and effectiveness of ciliary action can be controlled by temperature and the reaction is reversible within limits. The effectiveness and frequency of the beats were greatest between 18C and 33C in the nasal mucosa of rabbits and human beings. In the mucosa of frogs 13C was the most favorable temperature for ciliary action. When the temperature was elevated or reduced, there occurred a diminution of activity which became more and more striking with the reduction or elevation of the temperature until the ciliary motion finally ceased.

The action of ions on ciliary movement is interesting. The effectiveness of ciliary activity is greatest at pH 7.8. A little alkaline or especially a little acid reaction nullifies the effect. The experimental figure of pH 7.8, which causes most vigorous motion of the cilia, corresponds closely to pH found in normal human secretion.

The concentration of salt solution in which the specimen of mucosa was kept can affect the frequency of beat. Rabbit cilia remain active for a long time in a solution of from 0.6% to 0.9% of sodium chloride at room temperature. In a 0.6% solution of NaCl, ciliary action entirely ceases.

There is no connection between ciliary movement and the autonomic nervous system.

HARA, Los Angeles

News and Comment

ANNOUNCEMENTS

International Congress on Modern Educational Treatment of Deafness.—The International Congress on the Modern Educational Treatment of Deafness will meet at Manchester University, Manchester, England, from July 15 to 23, 1958.

The Congress is being organized with the approval and support of the Council of the University, with financial help from the Ministry of Education, the National College of Teachers of the Deaf, and the Rathbone Trustees, and with the cooperation of the British Council and the Alexander Graham Bell Association for the Deaf, Inc., Washington, D. C.

The Congress is being planned to bring together educators, audiologists, otolaryngologists, pediatricians, medical officers, and educational psychologists to consider and discuss the significance, to the future development of their work, of the results of modern research on problems of deafness in childhood.

It is hoped that the Congress will provide an opportunity to gather together, and to consider, all possible information about the ascertainment, assessment, and treatment of deafness in children of preschool age, and provision of education for boys and girls with impaired hearing through the work of the medical services, audiology clinics, special schools and classes, and placement in ordinary schools.

It is intended to arrange to hold a number of plenary sessions as well as a number of sectional meetings in which members of the Congress will be able to meet in small groups to study and discuss specific topics.

Arrangements are being made to offer accommodation to members of the Congress in University Halls of Residence. The cost of this type of accommodation is expected to be about £1.2.0 d. per day, including bed, breakfast, and dinner. The University Refectory will be available for meals during each day.

For those who wish to do so, arrangements will be made to visit audiology clinics and special schools in London, Oxford, and other places. It is also planned to organize tours to places of interest.

Forms of application for membership of the Congress will be supplied upon request to Prof. A. W. G. Ewing, Department of Education of the Deaf, The University, Manchester 13, England.

Seventh International Congress of Bronchoesophagology.—The Seventh International Congress of Bronchoesophagology will be held in Kyoto, Japan, Sept. 12-14, 1958. Persons wishing to present a scientific paper before the Congress should submit a summary, containing not more than 800 words, to the Director, Prof. Mituharu Goto, M.D., Otorhinolaryngological Clinic, Kyoto University Hospital, Kyoto, Japan. This summary must be in the hands of the Director not later than April 30, 1958.

Books

Oral Medicine. Third Edition. By Lester W. Burket D.D.S., M.D., Professor of Oral Medicine and Dean, School of Dentistry, University of Pennsylvania; Professor of Oral Medicine, Graduate School of Medicine, University of Pennsylvania. Price, $14.00. Pp. 558, with 351 illustrations. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1957.

This third edition has brought up to date an excellent book which correlates oral diseases with systemic diseases. Its intent is to bring into closer relation the dentist and the physician by presenting many medical conditions which may have local manifestations involving the mouth and teeth.

The first section deals with general problems as related to medicine and dentistry and includes history taking. The next section deals with local conditions involving the gingiva. The third section deals with the tongue and dermatologic diseases of dental interest. Section 4 deals with diseases of the organ system, such as respiratory, cardiovascular, gastrointestinal, urogenital, reproductive, endocrine, blood, bones and joints, nerves and the nervous system. Section 5 deals with nutritional deficiencies. Succeeding sections discuss specific infectious granulomas, focal infection, dental pediatrics, and gerodontics, and occupational hazards. Dr. S. Gordon Castigliano has revised his chapter on oral cancer. Section 10 is concerned with the various laboratory aids in diagnosis.

In addition, there is an excellent color atlas and finally a regional diagnostic index.

This book represents a most comprehensive description of oral pathology with an intimate relation to medical diseases. It should find a place on the desk of every dentist as well as the general practitioner and otolaryngologist.

Topographische Ausdeutung der Bronchien im Röntgenbild. By Claus Esser, M.D., Priv.-Doz., Mainz. Price, Cloth, $13.50. Pp. 210, with 89 illustrations. Georg Thieme Verlag Herdweg 63 (14a), Stuttgart. (American agent—Grune & Stratton, Inc., 381 4th Ave., New York 16), 1957.

This second edition deals with an intimate correlation of the anatomy and roentgen diagnosis of the bronchi. The first chapter deals with the anatomy of the lungs, each lobe and the various bronchi supplying each segment. Chapter 2 presents methods of roentgen examination to emphasize and localize the individual bronchi. This includes demonstrations of body sections. Chapter 3 is devoted to a more critical anatomical and roentgenographic presentation not only of each bronchus but also the bronchioceles. The relation of the vascular system to the entire bronchial tree is illustrated. The text is amplified by photographs of anatomical sections, roentgenograms, tomograms, and line drawings.

This book is presented with extreme attention to detail and should prove of great value to every roentgenologist.

Der Hirnabszess. By G. Weber, M.D., Priv.-Doz. for Neurosurgery, Neurosurgical Clinic, Zurich. Price, Paper, D. M. 29.70 ($7.10). Pp. 185. Georg Thieme Verlag, Herdweg 63 (14a), Stuttgart. (American agent—Grune & Stratton, Inc., 381 4th Ave., New York 16), 1957.

This monograph is a comprehensive discussion of intracranial complications with detailed emphasis on brain abscess.

The first section deals with the various intracranial complications following skull injuries. There is an evaluation of these complications, including incidence, pathogeneses, and bacteriology. The second section deals with infections of rhinologic or otitic origin. Subsequent sections are concerned with symptomatology and therapy of intracranial suppurations in association with osteomyelitis of the skull, thrombosis of the intracranial sinuses and cerebral veins, subdural infections, and suppurative leptomeningitis. The last section deals with brain abscess, its pathogenesis, pathologic anatomy, symptomatology, treatment, and prognosis. This entire text is replete with case reports and references with comparative tables of many other investigators. This monogram brings out the numerous problems involved and brings to date the changes in pathology and therapy as they are influenced by antibiotic therapy. This is an excellent treatise for the otologist and the neurosurgeon.

The Early Diagnosis and Treatment of Acoustic Nerve Tumors. By J. Lawrence Pool, M.D., Professor of Neurosurgery, Columbia University College of Physicians and Surgeons, and Arthur A. Pava, M.D., Assistant Neurosurgery, Wesson Memorial Hospital, Springfield, Mass. Price, $5.50. Pp. 161, with 33 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

This monograph presents a succinct and comprehensive discussion of the authors' experience in the surgical management and results of total and subtotal removal of acoustic nerve tumors.

Stress is placed on early diagnosis and the close cooperation of the otolaryngologist, neurologist, radiologist, internist, and neurosurgeon so that more successful long-term postoperative results and even the preservation of hearing is achieved.

The monograph is based primarily on the authors' experience derived from 135 cases of acoustic nerve tumors. Their results are correlated with those of other neurosurgeons, such as Henschen, Cushing, Revilla, Edwards and Paterson, and Olivecrona.

There is a brief discussion of histogenesis, pathology, and related data such as age, sex, and laterabity. The various symptoms are coordinated. A detailed description of the various diagnostic procedures is presented. These include x-ray, encephalography, pneumoencephalography, ventriculography, arteriography, EEG., cerebrospinal fluid, audiometric and caloric tests.

A group of atypical cases are presented.

The various operative procedures are discussed with an evaluation of each and the problems encountered under varying circumstances. The morbidity is presented. There is also a chapter on facial nerve anastomosis.

This monograph should be of great help to the otologist, the neurosurgeon, and the internist.

The Rehabilitation of Speech. Third Edition. By Robert West, Merle Ansberry, and Anna Carr. Price, $7.50. Pp. 676, with 59 illustrations and charts (also tables, lists, and glossary). Harper & Brothers, 49 E. 33rd St., New York 16, 1957.

This is the third edition of the Rehabilitation of Speech, which was first published in 1937. The book is organized into two major parts: Book One, Pathology of Speech and the Rationale of Its Rehabilitation; Book Two, Remedial Principles.

The organization of this book results in the inclusions of several chapters not ordinarily found in textbooks on speech therapy—at least not as subjects to be treated separately. The reference is to chapters entitled Secondary Disorders of Speech, Undesirable Concomitants of Speech, Mongolism, and Remedial Procedures for Children. This reviewer found the classification of speech disorders, on page 269, cumbersome. It will probably not be very helpful to a new student or lay reader.

Perhaps the unique feature of this book, which appears in Chapter VI, is a series of 15 pictures designed to show the actual position of the organs of articulation during the enunciation of various speech sounds. These pictures are described as "reproductions of single frames of a motion picture study of the articulatory positions and movements of a patient in whose case pathology of the face had resulted in such removal of structures and tissues as to afford a window into the mouth and pharynx." These pictures are accompanied by a series of line drawings of the sagittal section of the head and neck, and a thorough discussion of the interpretation of both drawings and pictures. The feature of the book should be helpful to readers who have little knowledge of phonetics.

The chapter on dysphonias in the first part of the book contains an extensive analysis of various types of voice disorders, while the later chapter on remedial procedures presents a rather brief discussion of standard methods.

Chapter XXIII describes a number of the standard tests of acuity and perception of hearing employed by audiologists, and a brief discussion of methods employed in auditory training, speech reading, speech correction, and hearing conservation.

The book ends with an extensive glossary and a series of five appendices, covering Code of Ethics of the American Speech and Hearing Association, Clinical Certification, Requirements of the American Speech and Hearing Association, Minimum Requirements for Hearing Program Offering Guidance in Selection of Hearing Aids, Tests of Hearing, and Tests of Articulation.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.
Place: Nova Scotian Hotel, Halifax, N. S. Time: June 9-11, 1958.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 12-14, 1958.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. Henry M. Scheer, 522 W. End Ave., New York 24.
Secretary: Dr. Louis Savitt, 3402 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: Aug. 12, 1958.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Alden Miller, 500 S. Lucas Ave., Los Angeles.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco. Time: June 23-27, 1958.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Broncho-Esophagological Association

President: Dr. Walter Hoover, 605 Commonwealth Building, Boston.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

American Laryngological Association

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco. Time: May 19-20, 1958.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, October, 1958.
Annual Meeting: Chicago, October, 1958.

American Otological Society, Inc.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: Fairmont Hotel, San Francisco. Time: May 17-18, 1958.
Place: The Homestead, Hot Springs, Va. Time: 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Facial Plastic Surgery

President: Dr. Irvin J. Fine, 505 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Annual Spring Meeting: Columbus, Ohio, March 10-11, 1958.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.

Otosclerosis Study Group

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

Society of Military Otolaryngologists

President: Capt. William C. Livingood, U. S. N. (MC).
Secretary-Treasurer: Lt. Col. Stanley H. Bear, USAF(MC), 3810th USAF Hospital, Maxwell AFB, Alabama.

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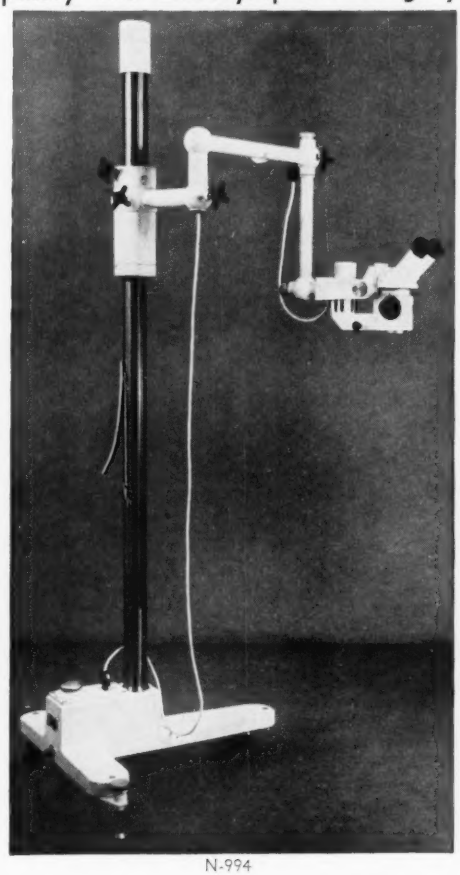

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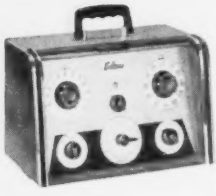

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